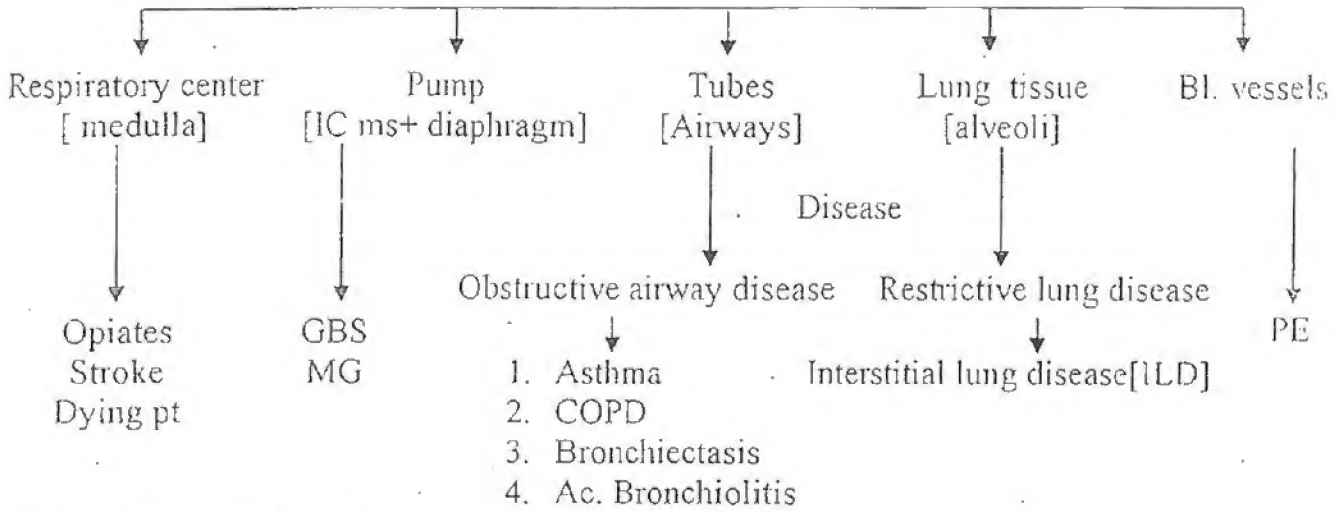
Respiratory Medicine

Importance of studying respiratory system:

- Asthma is the most common ch. disorder in children
- COPD is the 4th leading cause of death
- Lung cancer is the most common cause for cancer death

Respiratory system structure



- IC ms: Intercostal muscles
- GBS: Gillian-Bare-syndrome
- MG: Myasthenia gravis
- COPD: Chronic obstructive airway disease
- PE: Pulmonary emoblism

Pulmonary physiology

The main function of the lungs is gas exchange and for this it needs:

- Adequate ventilation of the alveoli [Open tubes & working pumps]
- Adequate perfusion of the alveoli [Open Bl. Vessels]
- Matching between ventilation (V) and perfusion (Q)
- Adequate diffusion of gases bw alveoli & bl. capillaries [normal alveolar wall]

Symptoms of respiratory diseases

Dyspnea | Cough | Wheeze | Chest pain | Sputum | Hemoptysis

Dyspnea

Definition: awareness of breathing

Causes:

1. All Respiratory disease
2. All Cardiac disease [by causing HF]
3. Anemia
4. Obesity
5. Psychogenic

DDX of acute dyspnea:

1. Asthma
2. Ac. Exacerbation COPD
3. Pneumothorax
4. Pulmonary embolism
5. Ac. Pulm. Edema [LVF]

Special types of breathlessness:

- **Orthopnea:** it is breathlessness on lying down [occurs in Heart failure, and diaphragmatic paralysis]
- **Trepopnea**: breathlessness on lying on the side [occurs in Heart failure]
- **Platypnea**: breathlessness on standing [occurs in hepatopulmonary syndrome due to intrapulmonary shunts]

Cough

- Cough is the most common symptom of respiratory disease

Pathophysiology: Cough receptors are found in the epithelium of the upper & lower respiratory tracts which when stimulated [infection, smoke, tumor] an impulse goes to the Cough center in the medulla leading to Cough

- Causes of Acute cough (< 3 weeks):
  1. Viral infections of the upper respiratory tract are the most common cause.
  2. Allergic rhinitis.
  3. Pneumonia.
  4. Acute heart failure
- Causes of Ch. cough (> 3 wks) in smokers:
  - Ch. bronchitis is the most common cause of ch. cough
- Causes of Ch. cough (> 3 wks) in non-smokers:

  - Common causes
  1. Asthma
  2. GERD
  3. Post-nasal drip syndrome
  4. ACEI
  5. TB

  - Less common causes:
  1. Bronchiectasis
  2. Interstitial lung diseases
  3. Lung cancer

Sputum

Correlation between Sputum and Disease	
Mucoid (Clear)	Ch. Bronchitis
Frothy (Clear or Pink)	Pulmonary edema
Purulent (Yellow → Green)	Infection
Bloody	TB, Infection, CA, Pulm. Embolism
• Yellow or Green sputum may occur in Asthma or Aspergillosis in absence of bacterial Infection • Large amounts of sputum mainly in the morning is characteristic for Bronchiectasis	

Wheeze

Definition: musical sound occurs in expiration due to the passage of air through narrowed bronchial airways.

Causes = DDx:

1. Obstructive airway disease [Asthma, COPD, Bronchiectasis]
2. Heart failure [Cardiac asthma]
3. Pneumonia

Chest pain

Chest pain in respiratory diseases occurs when the parietal pleura is involved and is characterized by that it is:

- Sharp
- Lateralized
- ↑ with inspiration

Respiratory causes of chest pain:

- Pneumothorax
- Pulmonary embolism → Pulmonary infarction
- Pneumonia
- Malignancy

Hemoptysis

Definition: Coughing up of blood.

DDx:

1. TB
2. Lung CA
3. Bronchiectasis
4. URTI & Pneumonia
5. Pulmonary embolism
6. Mitral stenosis
7. Blood disease

Respiratory system examination

Inspection

1. 5S: Symmetry, Swelling, Scar, Superficial veins, Skin discoloration.
   - Superficial veins may occur in superior vena cave obstruction.
   - Scar [surgical, or pleural tap]
2. Count the respiratory rate.
3. Deformities:

   - Chest should be symmetrical
     - Unilateral localized abnormality:
       - Bulge [Tumor, PTx, PE]
       - Retraction [Fibrosis, Collapse]
       - To know whether it is a bulge or retraction ask the pt to breath the part that moves with respiration is the normal and the other is the abnormal

   - Normally AP diameter is larger Transverse diameter [7:5 ratio]
     - **Barrel chest**: occurs when Transverse diameter is > AP diameter.
       Other Signs of hyperinflation: The ribs become more horizontal & sternal angle becomes more prominent & substernal angle becomes obtuse.
       Causes: Ch. obstructive disease [Ch. asthma, Emphysema, Bronchiectasis]

   - Other deformities:
     - **Pigeon chest** = Pectus carinatum: forward projection of lower sternum *Causes*: Congenital. Asthma during childhood. Rickets
     - **Funnel chest** = Pectus excavatum: indentation of lower sternum. [Congenital]
     - Kyphosis: backward curvature of the thoracic spine.
     - Scoliosis: lateral curvature of the thoracic spine.

4. Use of accessory muscles: Sternocleidomastoid

Palpation

1. Chest expansion:
   - Done at 3 levels from front or back to test all lung zones [done at same sites of percussion and auscultation] (see below)
2. Trachea central or not [First sit the patient] to test for upper mediastinal deviation
3. Apex beat [deviated or not] to test for lower mediastinum
4. TVF: Tactile Vocal Fremitus: palpable vibration of chest wall due to from speech
   - ↑ only in consolidation, or collapse with patent bronchus
   - ↓ **or absent fremitus**: Excess air in lungs[Emphysema, COPD and Asthma] Pleural disease [thickening or effusion or pneumothorax] and Collapse.

Percussion

- Sites:
  - **Anteriorly**:
    1. Directly on the clavicle
    2. On the 2nd &4th intercostal space at midclavicular line
    3. On the 6th intercostal space in anterior axillary line
  - **Laterally** on the axilla [For middle lobe in the Rt lung & Lingula in the Lt lung]
  - **Posteriorly**:
    1. When percussing for the chest from the back ask the pt to fold his arm.
    2. Apex
    3. On the 3rd 6th & 9th intercostal spaces & avoid precussing on the scapula
- Type of sound:
  - **Hyperresonance**: Emphysema, Pneumothorax.
  - **Resonant**: Normal
  - **Dullness:** Consolidation or Collapse
  - **Stony Dull:** Pleural effusion

Auscultation

- On Auscultation you have to comment on 4 things:
  1. **Air entry**: Normal, Reduced, or Absent. [Never ↑]
  2. **Type of breathing**
     - **Vesicular** – continuous and inspiration > expiration [Normal]
     - **Bronchovesicular** – vesicular with prolonged expiration [Obstructive airway diseases]
     - **Bronchial** - interrupted and inspiration = expiration [Consolidation]
  3. **Added sounds**
     - **Crepitations** =Crackles = Rales
     - **Rhonchi**: musical sound due to airway obstruction and heard mainly during expiration occurs in: Obstructive airway diseases. [Asthma, COPD, Bronchiectasis], May occur in pneumonia.
     - **Pleural friction rub** [occurs in pleurisy]: rubbing sound with late inspiration and early expiration.
  4. **Vocal resonance**: audible vibration of chest wall from speech. [changes as TVF].
- Especial test for consolidation:
  - **Whispered pectoriloquy**: Whispering of sound [44]is heard as load sound in area of consolidation
  - **Egophony**: whispering E is heard A– lung consolidation

Crepitations Cause	Mechanism	Effect of cough
Bronchiectasis (Coarse)	Due to passage of air through Bronchial secretions	↓ or disappear
Chronic bronchitis		
Pneumonia		
Pulmonary edema (Fine)		
ILD [Fibrosing alveolitis] (Fine)	Due to sudden opening of the collapsed alveoli at the end of the inspiration	No effect

To differentiate between ILD & other causes of crepitation ask the patient to cough and if crepitations disappears then it is not due to ILD

	Examination	Asthma	Ch. Bronchi	Emphysema	Bronchiectasis	ILD
	Inspection	± Barrel	± Barrel	Barrel	± Barrel	-
Palpa	Expansion	↓ Bilaterally	↓ Bilaterally	↓ Bilaterally	↓ Bilaterally	↓ Bilaterally
Palpa	Deviation	No	No	No	No	No
Palpa	TVF	N or ↓	N or ↓	N or ↓	N or ↓	N
	Percussion	Resonant	Resonant	Hyperresonant	Resonant	Resonant
Auscultation	Air-entry	↓ Bilaterally	↓ Bilaterally	↓ Bilaterally	↓ Bilaterally	N
Auscultation	Type breath	Vesicular	Vesicular	Vesicular	Vesicular	Vesicular
Auscultation	Added sound	Wheeze	Wheeze + crepitation	± Wheeze	Wheeze + Coarse crepitation	Fine crepitation
Auscultation	VR	N or ↓	N or ↓	N or ↓	N or ↓	N

	Examination	Pneumothorax	Pleu. Effusion	Collapse	Consolidation
	Inspection	± bulge	± bulge	± retraction	-
Palpa	Expansion	↓ Unilaterally	↓ Unilaterally	↓ Unilaterally	↓ Unilaterally
Palpa	Deviation	Away	Away	Toward	No
Palpa	TVF	↓	↓	↓	↑
	Percussion	Hyperresonant	Stony dull	Dull	Dull
Auscultation	Air-entry	↓ or absent Unilaterally			
Auscultation	Type breath	Vesicular	Vesicular	Vesicular	Bronchial
Auscultation	Added sound	-	-	-	Crepitation
Auscultation	VR	↓	↓	↓	↑

Investigation of respiratory system

- Chest-X-ray [CXR]
- Arterial blood gas [ABG]
- Pulmonary function test [PFT]

Pulmonary function tests (PFT)

➢ *Lung volumes measurement*

❖ *Spirometry can measure:* [Spirometry paper is called vitalograph]

- TV (Tidal volume): the volume of air that is inspired & expired during rest.
- VC (Vital capacity): the volume of air that can be expired after full inspiration.
- **FEV1** (Forced expiratory volume at the first second): it is the amount of air that you can forcibly expire in one second. Normally **> 80% of predicted**.
  - ↓ of FEV1 < 80% of predicted occurs in obstructive OR restrictive lung diseases
- **FVC** (Forced vital capacity): it is the amount of air that can be <u>forcefully</u> expired after full inspiration.
- **FEV_1 / FVC:** This is the ratio of FEV 1 to FVC. Normally **> 70%.**
  - ↓ FEV_1 / FVC < 70% occurs in obstructive airway diseases.

❖ *Helium dilution or body plethysmography can measure :*

- FRC (Functional residual capacity): the volume of air that remains in the lung at the end of a normal expiration.
- **RV (Residual volume)**: it is the amount that remains in lung and cannot be expelled after forceful expiration.
- **TLC (Total lung capacity)**: it is the total amount of air in the lungs.
- RV/TLC = 20-25% in normal but in emphysema it is > 35%.

$$TLC = RV + VC$$

According to PFT lung diseases are divided into Obstructive or Restrictive.
In all obstructive airway diseases the obstruction is more on expiration → trapping of air → ↑ in RV & TLC.

	TLC	RV	VC	FEV1	FEV1/FVC
Obstructive	↑	↑	↓	↓	↓
Restrictive	↓	↓	↓	↓	Normal

- **Peak flow meter**
  - It measures the peak flow rate (**PEFR**).
  - PEF is the maximum rate of airflow during a sudden forced expiration from a position of full inspiration.
  - It is a cheap and portable but it is **useful only in obstructive disease.** [↓ in the presence of obstruction].
  - Normally **> 80% of predicted.** [Correlates well with FEV1]
  - PEFR is used to monitor asthma patients.

- Diffusion capacity for carbon monoxide (DL_{CO}):
  - In this test, a small concentration of carbon monoxide (0.3%) is inhaled in a single breath that is held for ~10 seconds.
  - The value obtained for DL_{CO} depends on
    1. The alveolar-capillary surface area→ ↓ in **Emphysema**
    2. Pulmonary capillary blood volume → ↓ in pulmonary embolism, & vasculitis [e.g. Wegner's granulomatosis]
    3. Thickness of the alveolar-capillary membrane → ↓ in **ILD**
    4. The patient's hemoglobin level will affect the measurement.

Uses of PFT

1. To detect Type of lung disease & The extent of abnormality & disability
2. To detect The response to therapy
3. To detect The progression of the disease
4. For Pre-operative assessment

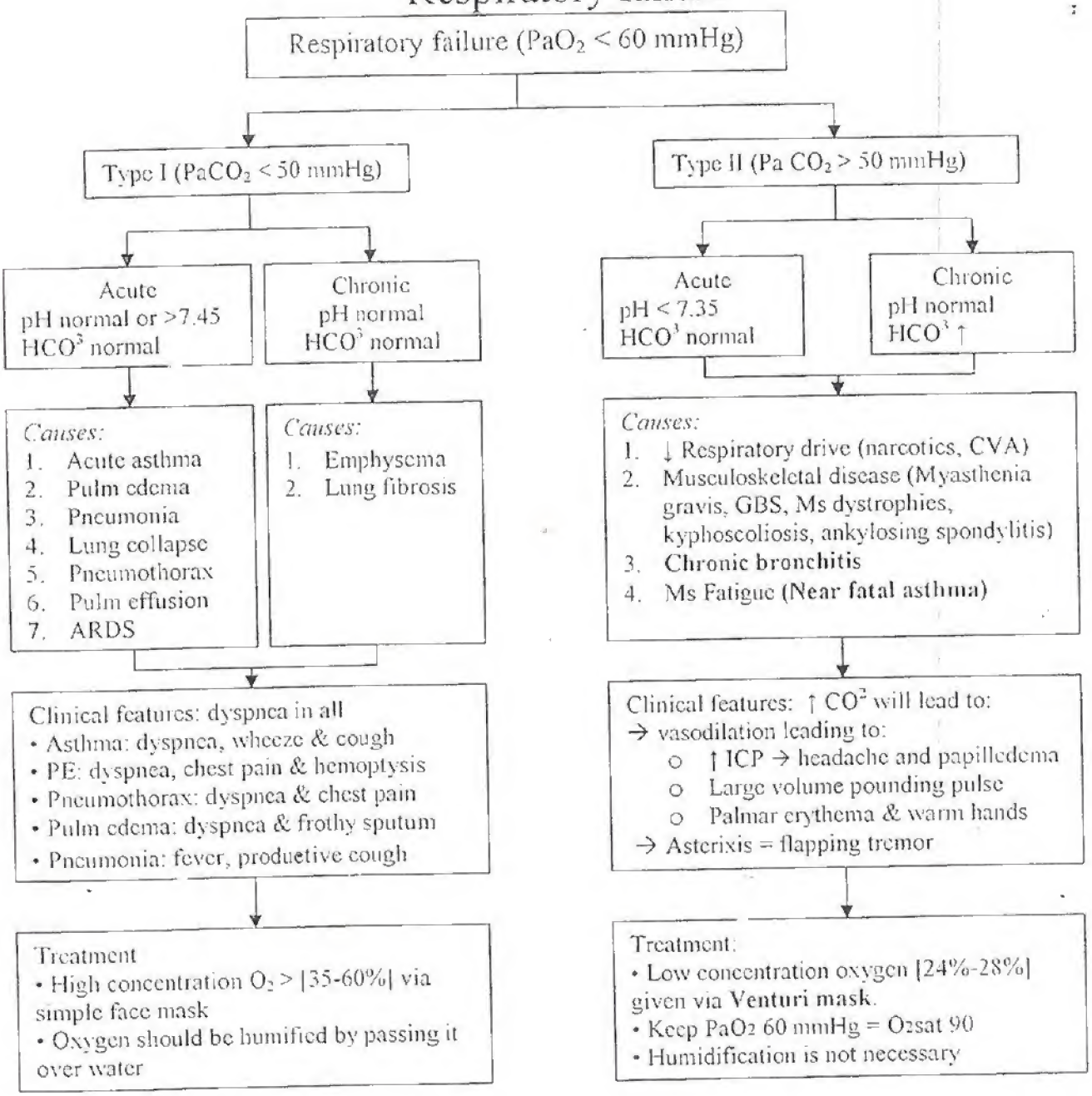
Limitations of PFT

1. Cannot reveal disease until certain extent of disease
2. Cannot provide anatomical diagnosis

ABG (Arterial blood gas)

Normal values	Abnormalities	
pH = 7.35 – 7.45	< <7.35 = **Acidosis**	>7.45 = **Alkalosis**
PO2 = 80 – 100 mmHg	< 80 mmHg → hypoxemia < 60 mmHg → respiratory failure	
PCO2 = 35 – 45 mmHg	< 35 = Hyperventilation > 45 = Hypoventilation > 50 = Respiratory failure	

Respiratory failure



Asthma

Definition

Asthma is chronic inflammatory airway disease, with hyperresponsiveness to a variety of stimuli leading to narrowing of the lower airway, the narrowing is **Reversible** either spontaneously or by medication. Clinically the condition **Episodic** and characterized by the **Triad of dyspnea, cough, and wheeze**.

Epidemiology

- Asthma is the most common chronic disease of childhood
- **40% of** pts with Atopic dermatitis have asthma.

Age: more in **children**
Gender → in children its more in **Boys**
→ in adults more in **Females**
Race: more in Black.

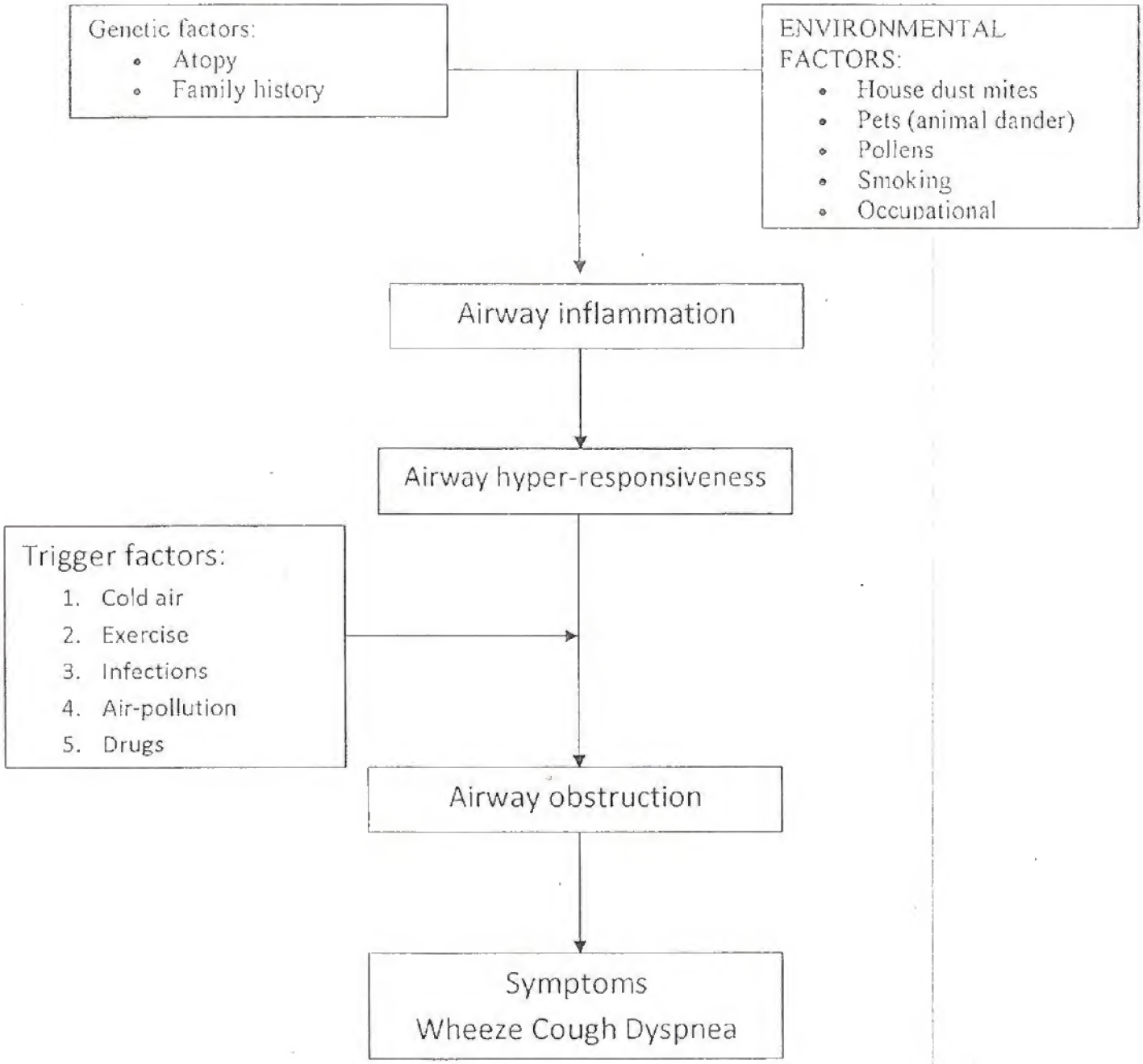
Etiology

Abnormal genes [Atopy genes] **+ Environment** [Allergen] + ? → ASTHMA

- Factors ↑ the risk of developing asthma:
  - Super hygiene hypothesis
  - **Maternal smoking** during pregnancy.
  - Obesity
- Protective factors:
  - Milk fat
  - Antioxidants [vitamin E and selenium]

Triggering = Inciting factors: [7]

1. Allergen → Dust mite
   → Pets
   → Pollens
2. Drugs: Most important are **Asprin** and B-adrenergic antagonist
3. Occupation [symptoms are related to work]
4. Viral infections most commonly RSV and Parainfluenza in children & rhinovirus and influenza in adults[NOT bacterial]
5. Exercise [exercise provokes asthma within minutes that resolves within 1 h].
6. Emotional stress
7. Exposure to cold air



Classifications of asthma:

- **Extrinsic asthma**: occurs in children, related to atopy, it is triggered by allergens [most commonly dust mite], usually seasonal, often remits by teenage.
- **Intrinsic asthma:** late onset asthma, related to smoking, no atopy.
- **Occupational asthma:** related to work place asthma

Clinical picture

Symptoms

Asthma = Episodes of Wheeze + Cough + Dyspnea

- Diurnal variation:
  - **Morning dipping:** symptoms are worse early in the morning
  - **Nocturnal asthma:** symptoms prevent pt from sleep
  - Most of pt have symptoms more at night
- The pt may have yellow sputum due to eosinophilia
- Some pt have only cough and called [cough-variant asthma]
- Rarely pneumothorax occurs

Aspirin-Sensitive Asthma **[Samter's Triad] or [Widal's Syndrome]**

1- Rhinosinusitis
2- Recurrent Nasal polyps [ask about them in Hx]
3- Asthma that is exacerbated by **aspirin**

More common in **female.**

Mechanism: aspirin inhibition of the cyclooxygenase pathway, with excess leukotriene production via the lipooxygenase pathway.

Signs

- Vital signs: increased RR, increased HR
- Pulsus Paradoxus: it is a poor guide for the severity of asthma.
- Respiratory system examination:
  - Signs of respiratory distress:
    1- Flaring of ala nasi
    2- Use of accessory respiratory ms [sternocleidomastoid]
    3- Suprasternal, Intercostal and Sucostal recessions

Resp. Ex.	Inspection	In pediatrics: Pigeon chest and Harrison's sulcus
	Palpation	↓ chest expansion bilat TVF equal bilat
	Percussion	Equal bilat
	Auscultation	↓ Air entry Vesicular breathing with prolonged expiration Rhonchi all over the chest

- Atalectasis or spontaneous pneumothorax rarely occur.

Investigation

- CXR: normal or shows Hyperinflation.
- ABG: (respiratory failure type I)
  - O_2 → hypoxia
  - CO_2 → reduced [if CO_2 is ↑ it is called life threatening asthma]
  - pH → respiratory alkalosis
- PFT: Obstructive pattern:
  - FEV1 < 80% of predicted
  - FEV1/FVC = < 70% of predicted
- PEFR: < 80% of predicted
- CBC → eosinophilia
- Sputum:
  - Churchman's spirals = mucous casts of the small airway.
  - Charcot laden crystals = breakdown products of eosinophils
- Metacholine/histamine challenge measures bronchial hyperresponsiveness. PC_{20} is the dose of agent provoking a 20% fall in FEV_1. Asthma is indicated by a PC_{20} of below 8 mg/ml. Normal subjects have a PC_{20} > 16 mg/ml.

Q. How to Diagnose Asthma?

Asthma is diagnosed by finding any of the following:

- Spirometry:
  1. FEV1 > 15% after a short acting beta 2 agonist
  2. FEV1 > 15% after a 14 day course of prednisolone 30mg
  3. FEV1 > 15% decrease following 6 minutes of exercise
- Peak flow meter:
  4. > 20% diurnal variation on > 3 days in a week for 2 weeks on peak expiratory flow diary

  {diurnal variation % = [(Highest – Lowest PEFR) / Highest PEFR] x 100}

Q. How to monitor Asthma?

By the use of **Peak Expiratory Flow Meter**

DDx of asthma:

1- COPD
2- GERD
3- Left ventricular Heart Failure

Management of Chronic asthma

Q. What are the Aims of Management of asthmatic pt?

1. Control of symptom → Pt can:
   - Attend school or job daily with normal daily.
   - Participate fully in the sport of their choice.
   - Sleep well without disturbance due to asthma.
2. Experience little or no adverse effect from therapy.
3. Prevent asthma exacerbation.

➢ Education of the pt:
- About asthma.
- How to use inhalers.
- How to use peak flow meter.

➢ Avoidance of precipitating factors

1. Allergen → Dust mite [↓ mattress in house]
   → Pets
   → Pollens
2. Drugs: Avoid Aspirin & β-blockers
3. Viral infections protection by annual influenza vaccine
4. Emotional stress
5. Exposure to cold air

➢ Drug treatment:
- **Bronchodilator drugs (relievers)** are used to relieve symptoms of bronchoconstriction.
- **Anti-inflammatory drugs (preventers)** treat the underlying chronic inflammatory present in asthma.

Bronchodilators

1. *β2-agonists*: (e.g. salbutamol, terbutaline) [Inhaler]
   - Mechanism: stimulate β-receptors → airway smooth-muscle relaxation
   - Onset of action: within 15 minutes.
   - Duration of action of 4–6 hours.
   - Side-effects: → Tremor.
     → Palpitations [tachycardia].
     → Muscle cramps.
2. *Long-acting* b2-*agonists*: (e.g. salmeterol, formoterol) [Inhaler]
   - Duration of action is >12 hours
   - useful in **controlling nocturnal symptoms**.

3. *Anti-cholinergic bronchodilators*: (e.g. ipratropium, oxitropium) [Inhaler]
   - Mechanism: blocking the bronchoconstrictor effect of vagal nerve.
   - Duration of action of about 4–6 hours.
   - Side-effects: may aggravating glaucoma.
   - Useful in young children and older patients.

4. *Theophyllines* [Aminophylline] (Oral or IV) [no inhaler]
   - Mechanism: phosphodiesterase enzyme inhibitor → ↑(cAMP)
   - Loading dose: 5mg/kg (very slowly) maintenance dose 0.5 mg/kg/hr.
   - Side-effects: → Nausea, vomiting,
     → Headache, convulsions
     → Tachycardia, arrhythmias

NOTE: Magnesium sulphate is smooth muscle relaxant used in acute severe asthma.

Anti-inflammatory drugs

1. *Inhaled corticosteroids* (e.g. beclometasone,budesonide, fluticasone)
   - Mechanism: inhibit the **airway inflammation (preventative treatment)**
   - **Compliance** is improved by using a twice-daily regimen every night and morning.
   - SE: → Oropharyngeal candidiasis [↓ by clean mouth with water after inhalation]
     → Hoarseness of the voice
     → ↓ adrenal function
     → ↑ osteoporosis

2. *Sodium cromoglycate* & Nedocromil ,
   - Mechanism: mast cell stabilizer. It is used in children to prevent exercise-induced asthma.

3. *Oral steroid treatment*:
   - Used for control exacerbations of asthma, for < 14 days and can be stopped suddenly.
   - Small number of pts need long term systemic prednisolone to control severe asthma. Steroids should not be stopped suddenly because of the risk of adrenal failure.

4. *Leukotriene receptor antagonists* (e.g. montelukast, zafirlukast)
   - Mechanism: block the effects of leukotrienes [metabolites of arachidonic acid]
   - Given orally in tablet form.

Stepped regime for management of asthma

Step 1 [mild intermittent asthma]:

- Inhaled Short-acting β_2 agonist for symptom relief.

Step 2 [regular preventer therapy]

- Inhaled Short-acting β_2 agonist for symptom relief.
- Low-dose inhaled corticosteroids
- Indication for Step 2 (see table) + if the Pt has an asthma exacerbation in the last 2 yrs

Step 3 [add on therapy]

- Inhaled Short-acting β_2 agonist for symptom relief +
  - High-dose inhaled corticosteroids
  - Low-dose inhaled corticosteroids + Long acting β_2 agonist

Step 4 [trial of one or more of the following]

- Leukotriene receptor antagonists [about 1/3rd of pts respond]. Trial for one month, and stop if no response.
- Theophylline
- Oral β_2 agonist.

Step 5

- Add oral steroid

Stepwise reduction is taken after the asthma has been stable for 3-6 month

Category	Clinical characteristics		FEV1	PEFR variability	Management
	Daytime Sx	Nighttime Sx			
Intermittent	≤2 days/wk	≤2 nights/mo	≥80%	<20%	Step1
Mild persistent	>2 days/wk but not daily	>2 nights/mo	≥80%	20–30%	Step 2
Moderate persistent	Daily	>1 night/wk	>60% – <80%	>30%	Step 3
Sever persistent	Daily	Frequent	≤60%	>30%	Step 4 or 5

➢ *Management of exercise induced asthma:*

- Warm up before exercise
- β-agonists or Sodium cromoglycate before exercise
- Leukotriene antagonist may be used

Management of Acute exacerbation of asthma

Severe asthma	Life threatening asthma	Near fatal asthma
Any of: 1. PEFR < 50% 2. RR > 25 3. HR > 110 4. Inability to complete sentence in one breath 5. Pulsus paradoxus	Any one of: 1. PEFR < 33% 2. Silent chest, or Cyanosis 3. Bradycardia or hypotension 4. Exhaustion, Confusion, Coma 5. ABG: ↓ O_2, Normal CO_2,	• ↑ $PaCO_2$ = Admission to ICU & Mechanical ventilation

- **Immediate management**
  - **Oxygen**: Highest concentration [60%]
  - **Bronchodilators: nebulised salbutamol** 5 mg repeat every 20 min if no improvement
  - **Steroids:** oral prednisolone 40 mg or hydrocortisone 100-200 mg 6-hourly IV

- If pt has life-threatening asthma add **Nebulised ipratropium** to nebulised β-agonist.
- If the pt doesn't respond → **IV Magnesium sulphate** (single dose only) – if no response → **IV aminophylline** if no response consider **IV salbutamol**. If no response then Do CXR to exclude pneumothorax
  Admission to ICU and mechanical ventilation
- Sedation must be avoided

History of pt with Asthma

MC: Wheeze: Onset, Duration, Frequency: of day symptoms, night symptoms. Course. Diurnal variation, Severity: sleep?. Precipitation factors [ALL 7], Relieving factor. H/o nasal polyps. Seasonal or perennial. Any associated symptoms?

ROS: GIT [Heart burn] and CVS [LVF] are important

PMH: when did the first attack occur [in adult it may during childhood]
Previous admissions due to asthma, & Admission to an ICU
Number of visits to ER.
Drugs used and their side effects.

FH: for the Atopy group

SH: MOST IMPORTANT (Ask about exposure to smoke, Ask about house ventilation, Exposure to pets, going to farm. Sport related activity, School Performance, Days missed from work).

Chronic Obstructive airway disease

Definitions

COPD is the presence of **Fixed** airways obstruction and it contain 2 main condition:

- **Chronic bronchitis** (clinical definition): the presence of productive cough for at least 3 successive months/yr for 2 successive years.

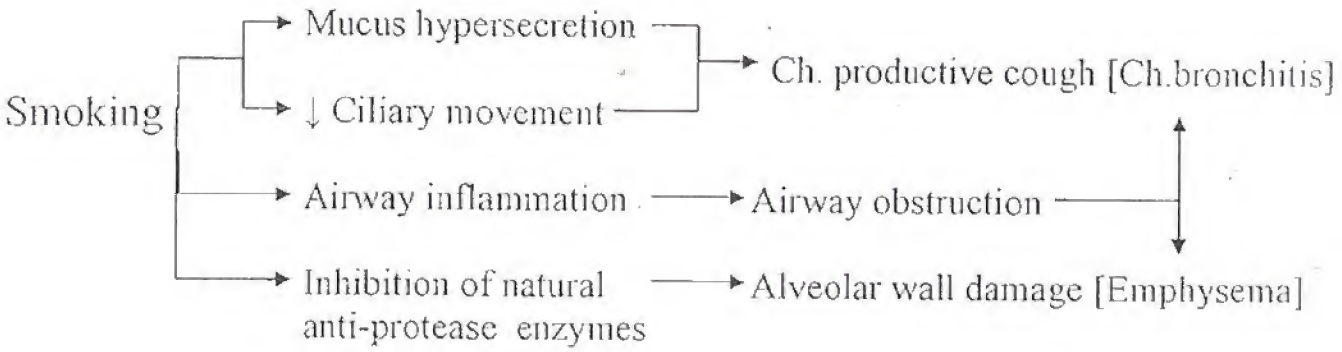
- **Emphysema** (pathological definition): it is permanent enlargement of the distal to terminal bronchioles due to destruction in the elastic alveolar wall.

Epidemiology

- It is the 4th leading cause of death in the world
- Age > 40 years
- Males > Females

Etiology

- Smoking [15% of smokers will develop COPD]
- It is unusual to develop COPD with < 10 pack years
- 1 pack year = 20 cigarettes/day/year



are cause of Emphysema is **α l-antitrypsin deficiency** (autosomal recessive):
In the body there are proteases (elastase) produced by lung neutrophils and they tend to digest the alveolar wall. Therefore there are antiproteases (α l-antitrypsin) produced by the liver to protect the lung.
The pt is < 40 years with **emphysema ± liver cirrhosis**
The emphysema occurring in this type is panacinar and affects mainly upper lobes
Unlike emphysema related to smoking which is centriacinar & affects lower lobes

Clinical picture

Symptoms → Dyspnea
Cough productive of whitish mucoid sputum
Wheeze

Signs

- Pt may show Pursed lips which help the pt breathing.
- Look for nicotine stain in the pt hands.
- Clubbing does **not** occur in COPD & if found look for malignancy.
- In pt with obstructive airway disease , the time for expiration decreases, resulting in progressive hyperinflation of the chest.
- Inward movement of the lower chest.

Resp. Ex.	Inspection	Barrel (hyperinflated) chest
	Palpation	↓ chest expansion bilat TVF normal or ↓ Impalpable apex beat
	Percussion	Hyperresonant in emphysema
	Auscultation	Rhonchi and crepitation in Ch. bronchitis

Difference bw Emphysema and Ch.bronchitis

In emphysema the pt is thin and breathless to maintain a normal $PaCO_2$
In ch. bronchitis the pt tolerates hypercapnia → LLE
→ 2ndry **polycythemia**
→ Pulm HTN → RVH→ RVF
Most commonly the pt has an overlap bw the 2 conditions.

	Emphysema **(Pink puffer)**	Chronic bronchitis **(Blue bloater)**
Built	Thin	Obese
Cyanosis	Absent	Prominent
Dyspnea	++	+
Hyperinflation	+++	+
Cor pulmonale	rarely	Often [↑JVP, LLE]
Resp. Failure	Type 1	Type 2

DDx of COPD:

1. Asthma
2. Bronchiectasis
3. TB
4. Heart failure

Investigation

- **Radiology**
  - **CXR:**

CXR in Emphysema:
1. Lung is more black
2. Ant ribs are more horizontal
3. Widening of intercostal spaces
4. The lung extend below → 6th rib ante
   → 10th rib post
5. Heart is tubular
6. Diaphragm is flat

  - **CT**: For Emphysema the best diagnostic method is CT scan.
  - In emphysema lung bullae may be seen on CXR, which if large can be mistaken for a pneumothorax due to the loss of lung markings. In this case do CT to differentiate between the two conditions.

- **ABG** Ch. bronchitis may → Resp. failure type II
  Emphysema may → Resp. failure type I

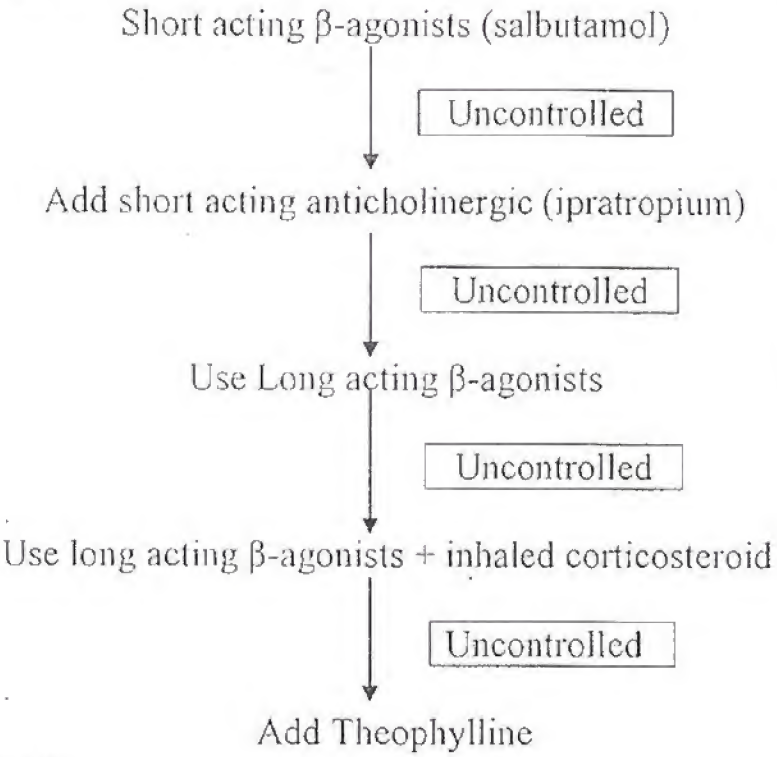
- **Pulse oxymetry** → hypoxia
- **PFT**: will show obstructive pattern with:
  - FEV_1 < 80% of predicted
  - FEV_1/FVC < 70%

  → Bronchodilator reversibility <15%
- DLCO: ↓ in emphysema

COPD severity	
Severity	**FEV_1**
Mild	50-80% predicted
Moderate	30-49% predicted
Severe	< 30% predicted

Management

- *Smoking cessation*: stops the progression of disease and ↓ **mortality**
- *Bronchodilators*



- *Corticosteroids*
  - Inhaled corticosteroids ↓ the frequency & severity of exacerbations; they are used in pts with severe disease with 2 or more exacerbations per year.
  - Oral corticosteroids: used **only** during acute exacerbation
- *Mucolytics:* for pt with productive cough
- *Oxygen therapy:*
  - Long-term domiciliary oxygen therapy (LTOT) ↓ **mortality**
  - Oxygen Rx ↓ the risk of developing Pulmn.HTN & 2ndry Polycythemia
  - Indication: Pt stopped smoking & PaO_2 <7.3 kPa.
  - The pts should use oxygen for at least 15 hrs/day.
  - Aim of Rx: is to ↑ the $\underline{PaO_2 \text{ to } 60 \text{ mmHg}}$ or $\underline{SaO_2 \text{ to } 90\%}$.
- *Pulmonary rehabilitation (exercise)*
- *Surgery*: Large bullae → considered bullectomy.
  Advanced disease → Lung transplant.
- *Vaccines*: Influenza vaccine annually and Pneumococcal vaccine

Acute exacerbation of COPD [ER]

Definition: an acute ↑ in symptoms ↓ lung function & health status in COPD pt.
During attack pt may develop Resp. Failure.
Causes:

1. Infection Bacterial [most common is Strep. pneumonia] or viral.
2. Arrhythmias
3. Pulm. Embolism
4. LVF

Treatment of acute exacerbation of COPD

1. ***Oxygen* therapy with concentration of 24to28% via Venturi mask.**

> • Physiologically ↑ CO_2 or ↓O_2 can stimulate the respiratory center.
> • In normal person the blood level of CO_2 drives respiration.
> • In pts with COPD **chronic** increased CO_2 leads to tolerance of the respiratory center to CO_2 and the respiratory center will depend on ↓ O_2 (hypoxia) to stimulate respiration (**Hypoxic Drive).**
> • Therefore in pts with COPD high dose oxygen can reparatory center depression → worsening of the pt condition.

2. **Bronchodilators:** Short-acting β-agonists (salbutamol) ± anticholinergics (ipatroprium)
3. **Corticosteroids:** given orally.
4. **Antibiotics** if sputum is purulent, pyrexial, ↑CRP, new changes on CXR.
5. **Heparin**
6. **Doxapram**: respiratory center stimulant. Used to ↑ RR (if < 20 breaths/min)
7. Mechanical ventilation if the pt didn't improve

Complications of ch. bronchitis

1. Resp. failure type II: ↑ CO_2 → vasodilation leading to:
   - ↑ ICP → headache and papilledema
   - Large volume pounding pulse
   - Warm hands & palmar erythema
   - Asterixis = flapping tremor
2. Pulmonary hypertension
3. Cor pulmonale:
   - Definition: Right ventricular hypertrophy± failure due to lung disease with normal Left ventricle.
   - RVF is suggested by finding ↑JVP, Hepatomegaly, and LLE.
4. Peptic ulcer.

Bronchiectasis

Definition: Irreversible abnormal dilatation of one or more bronchi, with chronic airway inflammation and obstruction.

Pathophysiology

The initial insult is infectious → damage the airways → abnormal anatomy → accumulation of secretion → secondary infection → ongoing inflammation with more airway damage.

Etiology

- Infections:
  - TB is the most common cause.
  - Post-pneumonia (whooping cough or measles)
  - Allergic bronchopulmonary aspergillosis [an immune reaction to aspergillus → bronchiectasis mainly in proximal airways of upper lobes].
- Congenital:
  - Cystic fibrosis
  - Congenital ciliary dysfunction:
    - **Kartagner's syndrome** (bronchiectasis, dextrocardia, ch sinusitis ± situs inversus)
    - **Young's syndrome** (bronchiectasis, sinusitis, and azoospermia)
  - Hypogammaglobulinemia
- Idiopathic

Localized bronchiectasis occurs beyond an obstructing bronchial lesion:

1. Enlarged hilar lymph nodes
2. Bronchial tumor
3. Foreign body

Clinical picture

- Symptoms
  - Constitutional symptoms: anorexia, malaise, fever, weight loss.
  - Dyspnea
  - Chronic Cough productive of large amount of highly viscous **purulent** sputum. The cough is worse in the **morning** and related to **posture**.
  - Halitosis
  - Wheeze
  - Hemoptysis [due to erosion of the bronchi]

- Signs
  - Clubbing of fingers
  - **Coarse** inspiratory & expiratory **crepitations** which ↓when pt is asked to cough [The cause of crepitation is the passage of air in bronchus filled with mucous]
  - Rhonchi.

Investigations

Laboratory

- Sputum microbiology [including for atypical organisms: acid-fast bacilli, & Aspergillus]
- **Saccharin test:** The time for saccharin to be tasted in the mouth after deposition on the nose. [normally < 20 min]
- Immunoglobulin levels

Radiology

- CXR may show:
  - Ring shadows and Tramlines [indicating thick airways]
  - Gloved finger appearance [consolidation around thick & dilated airways]
  - Honey-comb appearance [late]
- **High resolution CT chest is the best.**

PFT: obstructive pattern with incomplete reversibility [as COPD]

Treatment

- Physiotherapy (5-10 min) once or twice daily to keep the dilated bronchi empty of secretions.
- Bronchodilator for obstruction
- Antibiotics for infections
- Mucolytics for thick sputum
- Surgery : considered if bronchiectasis is unilateral & confined to a single lobe

Complications

- Respiratory failure
- Recurrent infection
- Pulm HT → cor pulmonale
- 2ndry pneumothorax
- Brain abscess
- Amyloidosis (AA type) resulting in nephrotic syndrome

Cystic fibrosis

Definition: It is an autosomal recessive disease characterized by dysfunction of the exocrine glands → obstructions in the lungs, pancreas, & GIT.

Epidemiology

- The most common fatal autosomal recessive disease in Caucasians.

Etiology a& pathogenesis

- The defective gene is found in the long arm of **chromosome 7**. And the abnormality is a deletion of the amino acid at position 508 in the protein.
- This gene produces a protein called cystic fibrosis transmembrane regulator (CFTR).
- Abnormal CFTR protein in Resp epithelial cell → Cl^- impermeability + ↑ Na^+ resorption → viscid mucus → obstruction exocrine glands.
- Abnormal CFTR protein in Skin epithelial cell → ↑ Na Cl conc. in sweat.

Clinical features

- Pulmonary manifestations
  - Obstruction→infection→Bronchiectasis
  - Predominant cause of infection is Pseudomonas aeruginosa & Staph. aureus.
  - Many pts develop sinusitis & nasal polyps.
  - Most patients die of respiratory failure.
- Non-pulmonary manifestations:
  - Clubbing
  - Intestine: **meconium ileus** .
  - Pancreas: malabsorption, DM.
  - Liver: fatty liver, biliary cirrhosis.
  - Genitourinary: Sterility in males. [> 95% of male pts are **azoospermic**]

Investigations

- PFT & CXR → As bronchiectasis
- **Sweat test:** Sweat sodium & chloride is > 60 mmol/l

Treatment

- As bronchiectasis
- In end-stage disease, bilateral lung transplantation

Interstitial lung diseases ILD

Definition

Interstitial lung disease (ILD)--or pulmonary fibrosis--refers to > 200 chronic lung disorders that are characterized by involvement of the tissue between lung alveoli (**the interstitium**) which is affected by inflammation → fibrosis.

All of these diseases are both under the a single heading because:

- All of them share similar Pathophysiology which is the involvement of the alveolo-capillary membrane (Blood-Air barrier) i.e. they involve Gas-Exchange.

  Blood-Air barrier → Alveolar epithelium
  → Capillary endothelium
  → Interstitial tissue

- All of these disorders have similar , *Clinical, & CXR manifestations.*

Etiology	
Known Cause	Unknown Cause
1. Pneumoconiosis 2. Hypersensitivity pneumonitis 3. Radiation 4. Drugs [Nitrofurantoin, Amiodarone, Gold-compounds, Bleomycin] 5. Aspiration pneumonia 6. ARDS	1. Idiopathic pulmonary fibrosis = Cryptogenic fibrosing alveolitis 2. Sarcoidosis 3. Amyloidosis 4. Autoimmune disease: • CTD (SLE, RA, Systemic sclerosis) • GIT [IBD, PBC] • Wegener's granulomatosis, Goodpasture's syndrome 5. Histiocytosis X

Clinical picture

History: Progressive dyspnea (usually Exertional) + Dry cough.

Important points to take in history:

- Past medical history → Collagen vascular disorder
- Social history → OCCUPATION, Exposure to pets, & smoking
- Drug history → Drugs may be the underlying cause

Examination: GE→ Look for Pulm. HTN & RVF

Resp. Ex.	Inspection	↓ chest expansion
	Palpation	↓ chest expansion bilat [use tape measure]
	Percussion	Resonant
	Auscultation	**Fine end-inspiratory crepitations that does not disappear after cough** (the mechanism of crepitations here is Sudden reopening of the collapsed alveoli)

Investigations

- **Laboratory test**
  - CBC→ lymphopenia in sarcoidosis; neutrophilia in hypersensitivity pneumonitis.
  - CRP and ESR
  - CTD screening [RF & ANA]
  - Ca^{2+} level may be increased in Sarcoidosis
- **Imaging**
  - CXR may be normal early in the disease
  - High-resolution CT (HRCT) is the **imaging investigation of choice** because more sensitive and specific, CXR & HRCT changes include:
    1. Reticular (crisscrossing lines)
    2. Nodular (lots of small dots)
    3. Rings or cysts (honeycombing in advanced disease)

Fibrosis mainly of upper zone	Fibrosis mainly of lower zones
1. Extrinsic allergic alveolitis	Other causes
2. Silicosis	
3. Sarcoidosis	
4. Ankylosing spondylitis	
5. Tuberculosis	

- **PFT**
  - Will show restrictive pattern (FEV1/FVC is > 70%) but TLC and RV are reduced.
  - DL_{CO} is **Decreased** [the most sensitive functional test].

BAL [Broncho-Alveolar Lavage]

- Idiopathic pulmonary fibrosis → Neutrophilia
- Sarcoidosis → Lymphocytosis [mainly CD4]
- Hypersensitivity pneumonitis → Lymphocytosis [mainly CD8]

- **Biopsy**
  - Lung biopsy is required when the diagnosis of interstitial lung disease is not clear and it may be transbronchial or open lung biopsy.
  - Lung biopsy is the gold standard for making the diagnosis of ILD.

Pathophysiology of ILD

ILD → TYPE I RESPIRATORY FAILURE

- Fibrosis of the lung → reduced compliance → ↑ work of breathing → dyspnea
- Thickening of the alveolo-capillary membrane → limitation of **oxygen diffusion** mainly on exercise but not at rest until the disease is so sever. [limitation of diffusion occurs to O_2 but not CO_2 **because** CO_2 is 20 times more diffusable than O_2]
- Hypoxia in the lung → vasoconstriction of pulmonary blood vessels –by time→ Pulmonary HT → right ventricular hypertrophy –by time→ RVF.

Idiopathic pulmonary fibrosis = Cryptogenic fibrosing alveolitis

Epidemiology

- Age > 50 yrs
- Gender: More common in males.
- More in smokers

Clinical picture

Symptoms

- Progressive dyspnea + Dry cough.
- Constitutional symptoms [Fatigue, Anorexia, Weight loss].

Signs

- See above
- Clubbing is common.

Investigation

- *Laboratory Findings*
  - ESR and LDH may be elevated.
  - Rheumatoid factor and ANA are found in 30-50% of pts.
  - ABG: Hypoxemia.
- *Imaging Studies*
  - CXR: Bilateral basal reticulonodular markings ± honeycombing.
  - High-resolution CT is the best imaging modality because it show abnormalities when CXR is normal (i.e. early in the disease).
- *PFTs:* Restrictive pattern. DLCO ↓.
- *BAL:* Neutrophilia ± Eosinophils
- *Histologic Findings:* Pts with typical clinical features & HRCT appearances do not require lung biopsy, but when diagnosis is not clear[biopsy confirms the diagnosis]

Treatment

Corticosteroid and immunosuppressive are used with best response is seen in pts with significant cellular infiltration and less fibrosis.
Note: no treatment has been shown to improve prognosis

Prognosis median survival of 3 years is typical and survival beyond 5 years unusual.

Note: rare acute form (**Hamman-Rich syndrome**) occurs with death within months

Sarcoidosis

Definition

A systemic disease of unknown etiology. It mainly affects the hilar lymph nodes & the lungs. It is characterized by the presence of noncaseating granulomas.

Epidemiology

- Age→ more common in young
- Gender→ more common in female
- Race→ more in black
- Smoking: ↓ the risk

Pathophysiology

- Unknown cause → activation of T cells → granuloma formation in the organ affected.
- Anergy of T-cell is found → depressed delayed hypersensitity reaction → negative TB testing
- Hypercalcemia occur in 10% of pts. The mechanism of $\uparrow Ca^{2+}$ is increased production of 1,25-dihydroxyvitamin D by the granuloma itself.

Clinical Manifestations

➢ Asymptomatic: In 30% of pt it present as asymptomatic hilar adenopathy.

➢ Acute sarcoidosis: 30 % of cases.

- *Lofgren's syndrome*: hilar adenopathy, erythema nodosum, acute arthritis & uveitis.
- *Heerfordt* **syndrome (uveoparotid fever)**: parotid enlargement, fever, anterior uveitis, facial nerve palsy.

Note: [**Mikulicz syndrome:** Enlargement of the parotid + Lacrimal glands may occur in sarcoidosis, tuberculosis or lymphoma]

➢ **Insidious onset Sarcoidosis**: 40% of cases.

Respiratory symptoms (most common) ± constitutional ± extrathoracic symptoms.

- *Constitutional symptoms*: fever, weight loss, anorexia, fatigue.
- *Resp: Lung is the* **most commonly involved organ**; 90% with sarcoidosis will have abnormal CXR sometime during course showing hilar lymphadenopathy. Fibrosis is seen in up to 20% of pts.
- *Skin:* **Erythema nodosum, & Lupus pernio** (indurate blue-purple shiny lesions on face, fingers, and knees).
- *Eye:* uveitis which may progress to **blindness.**
- *Hematology*: Splenomegaly and generalized lymphadenopathy may occur.

- *GIT*: asymptomatic disturbance in LFT.
- *Kidney*: nephrocalcinosis is rare, nephrolithiasis 2ndry to hypercalcemia .
- *Nervous system:* Chronic meningitis, space-occupying lesions, seizures.
- *CVS*: Conduction defect, pericarditis.
- *Musculoskeletal:* chronic non-erosive mono- or oligoarthritis of knee, ankle.

Investigations

- Laboratory tests:
  - CBC (Lymphopenia, anemia and thrombocytopenia may occur).
  - Ca^{2+} is ↑
  - ACE is ↑in 60% of pts
  - PPD test of TB is -ve.
- Radiology:
  - CXR
  - HRCT
- PFTs: Restrictive pattern • DLco ↓
- Bronchoscopy →Cobblestone appearance of the mucosa.
- Transbronchial biopsy shows non-caseating granulomas.
- Bronchoalveolar lavage: lymphocytosis with CD4 T-cell predominance

CXR changes in Sarcoidosis	
Stage I	BHL ONLY
Stage II	BHL & Parenchymal infiltrates
Stage III	Parenchymal infiltrates ONLY
Stage IV	Pulmonary fibrosis

Note: *Kviem test:* An intradermal injection of specially prepared tissue derived from the spleen of a known sarcoidosis pt is biopsied 4–6 weeks after injection. If noncaseating granulomas are seen, this is highly specific for sarcoidosis.

Treatment

- Most stage I cases resolve spontaneously.
- Indications for treatment:
  - Hypercalcemia
  - Involvement of the Kidneys, Eye, Heart, or CNS.
  - Lung infiltration or Progressive lung disease
- Glucocorticoids are main therapy. Methotrexate & Azathioprine are 2nd-line drugs.

Poor prognostic features: • Age > 40 • Black • Persistent Symptoms > 6 mo • Involvement > 3 organs • Lupus pernio • Stage III/IV chest X-ray

Pneumoconiosis (inorganic dust inhalation)

Definition: Inflammation & lung fibrosis due to inhalation of inorganic dust.

Coal Workers Pneumoconiosis

There are 3 types of coal worker pneumoconiosis based on CXR appearance:

- *Simple CWP*: CXR signs of simple CWP are small, irregular opacities (reticular pattern) that may progress to small, rounded opacities (nodular pattern), it has NO symptoms and NO effect on PFT, & NO progression after avoidance of exposure
- *Complicated CWP*: CXR appearance of nodules > 1 cm in diameter in upper lung fields ± cavitations; DLco is reduced, & pt has dyspnea & productive black cough (**melanoptysis**). It may progress after the avoidance of exposure.
- *Caplan's syndrome*: seropositive RA + Coal pneumoconiosis nodules.

Asbestos

The severity of asbestosis is related to the length of exposure. Unlike mesothelioma where even very limited exposure can cause disease. Asbestosis typically causes lower lobe fibrosis. Crocidolite (blue) asbestos is the most dangerous form.

Other features:

- Pleural effusion which is may be blood stained
- Pleural thickening
- Pleural plaques also seen (**not premalignant**)
- **Clubbing** in 40% of pts and it's a poor prognostic feature.

Diagnosis is based on H/O asbestos exposure + C/P of restrictive lung disease. Asbestos bodies may be identified in sputum or BAL and confirm asbestos exposure.

Silicosis

Silicosis is a risk factor for developing TB (silica is toxic to macrophages)

- Fibrosing lung disease affecting the upper lung zones
- **Egg-shell** calcification of hilar lymph nodes
- The disease continues after the cessation of exposure

Other pneumoconiosis	
Agents name	Disease name
Iron oxide	Siderosis
Tin oxide	Stannosis
Beryllium	Berylliosis
Talcosis	Talc

Extrinsic allergic alveolitis = Hypersensitivity pneumonitis (organic dust)

Definition: It is due to immune- mediated lung damage (type III & type IV HSR) due to a variety of inhaled organic particles. Note: Smoking: ↓ the risk

Causes

- Bird fanciers' lung (avian proteins)
- Farmers lung (spores of **Micropolyspora faeni**)
- Malt workers' lung (Aspergillus clavatus)
- Mushroom workers' lung (Thermophilic actinomycetes)
- Mouldy sugar cane exposure (**Bagassosis**)
- Budgerigar fanciers lung

Clinical picture
- Acute: occur 4-8 hrs after exposure, with dyspnea, dry cough, & fever.
- Chronic: dyspnea and dry cough and rarely clubbing.

Investigation

- CBC: neutrophilia & NO eosinophilia
- Serum precipitins (by ELISA) to offending antigen.
- CXR: Upper lobe fibrosis ± cavitations.
- PFT: restrictive pattern, DLco ↓
- BAL: lymphocytosis with $CD8^+$ T lymphocytes predominance

Treatment

- Stop exposure to the antigen is the FIRST line in management
- In acute cases prednisolone should be given

Cotton Dust (**Byssinosis**)
Unlike other organic dust, cotton dust inhalation → acute bronchiolitis [airflow obstruction] . More in smokers. Initially, symptoms occur after the weekend break (Monday fever) but later become continuous. CXR is usually free. Recovery follows stopping of exposure.

Miscellaneous conditions

Pulmonary Langerhans cell histiocytosis = Histiocytosis X

Definition: Due to unknown cause the lung.become infiltrated by histiocytes (Langerhans' cells). Histiocytosis may affect only the lung or it may be multisystemic.

Multi-systemic histiocytosis
- Letterer-Siwe syndrome: Acute dissemination of histiocytes
- Hand-Schueller-Christian syndrome: Chronic& presents with Triad of diabetes insipidus + Exophthalmos + Bony lesions

Epidemiology: Rare. Young Males [20-40 yrs]. Pt usually is a heavy smoker.

Clinical features: Exertional dyspnea and cough + Constitutional symptoms (Fever, Wt loss...) + **Pneumothorax** in ¼ of pts and may be the presenting feature.

Investigations
- CXR & HRCT: Reticulonodular shadows ± cavitations in Mid & Upper zones.
- PFTs: may normal, obstructive, or restricted. DLCO is ↓.
- Trans-bronchial biopsy or Surgical lung biopsy: Diagnostic.

Treatment: Smoking cessation + Oral corticosteroids for symptomatic disease.

Prognosis: median survival 12-13 yrs from diagnosis.

Goodpasture's disease

Definition: An autoimmune disease characterized by Linear deposition of IgG on the basement membranes of alveoli and glomeruli [Glomerulonephritis]

Epidemiology: Rare. Young Males [20-40 yrs]. More in smokers. HLA -DR2 association

Clinical features: Hemoptysis ± Hematuria.

Investigations
- CXR & HRCT: diffuse patchy shadows. Mainly mid and lower zones
- PFTs: Restrictive defect with ↑ DLCO if alveolar hemorrhage is present.
- Kidney: Urine analysis will show RBC casts.
- Renal biopsy: Linear IgG deposition detected by immunofluorescence technique

Treatment: High-dose steroids and cyclophosphamide.

Wegener's granulomatosis

Definition: Necrotizing vasculitis affecting small and medium size vessels, especially in the upper and lower respiratory tract and also the kidneys..

Epidemiology: Rare. Males = Females [40-50 yrs].

Clinical features: • URT (saddle nose, sinusitis, septal perforation) + LRT (cough, hemoptysis, dyspnea) + Renal disease (glomerulonephritis) + Constitutional symptoms

Investigations
- CXR & HRCT: **Cavitations** .
- c-ANCA +ve.
- Trans-bronchial biopsy or Surgical lung biopsy: Diagnostic.

Treatment: Smoking cessation + Oral corticosteroids for symptomatic disease.

Pneumonia

Definition

Pneumonia is an infection of pulmonary parenchyma and characterized by fever, cough, and dyspnea and new CXR changes.

Classifications

- Community-acquired
- Hospital-acquired or Nosocomial
- Recurrent pneumonia
- Aspiration pneumonia/Lung abscess
- Pneumonia in the immunocompromised host

Risk factors: • Smoking • Old age • Alcohol • DM • URTI • Corticosteroids

Community acquired pneumonia CAP

Epidemiology

- CAP is the commonest infectious cause of death & the 6th leading cause of death.

Clinical picture

Symptoms

➢ Typical pneumonia = Fever + Productive Cough + Dyspnea

- Constitutional symptoms [Fever, , anorexia, malaise, headache]
- Sputum is purulent [may be Rust-colored in *Streptococcus pneumoniae*]
- , Hemoptysis may occur
- Pleuritic chest pain if pleurisy occurs.
- Lower lobes pneumonia may cause upper abdominal pain

➢ Atypical pneumonia

- Pt have mainly constitutional symptoms and not respiratory symptoms.
- Cause: Mycoplasma (most common), Legionella, Chlamydia, Coxiella.

Signs

- ↑Respiratory rate ± Tachycardia
- A normal chest examination makes the diagnosis unlikely.

Resp. Ex.	Inspection →	↓ chest expansion of affected side.
	Palpation →	↓ chest expansion of affected side. No deviation
		TVF ↑
	Percussion →	Dull
	Auscultation →	↓ Air entry
		Bronchial breathing.
	↑ VR	Crepitations ± Rhonchi
	+veEgophony	Whispering pectoriloquy

Investigations

➢ *Laboratory*

- CBC: Leukocytosis (neutrophilia) [Counts of >20 or <4 indicate severe infection]
- ESR & CRP ↑
- Microbiological Ix → Sputum smear with Gram stain & Ziehl-Neelsen stains
  → Sputum culture
  → Blood culture

➢ *CXR:* Radiologically pneumonia may be:

- **Lobar pneumonia** homogeneous consolidation with air bronchogram of one or more lung lobes. Mainly caused by Streptococcus pneumoniae.
  The location of an consolidation on CXR & affected lobe:
  - Obscured right (R) costophrenic angle = right lower lobe
  - Obscured left (L) costophrenic angle = left lower lobe
  - Obscured Rt heart border = right middle lobe
  - Obscured Lt heart border = left upper lobe
- **Bronchopneumonia**: with patchy consolidation usually bilaterally affecting lower lobes.
- **Interstitial pneumonia**

➢ *Pulse-oxymetry*: Normal or Hypoxia

➢ *ABG*: Normal or RF type I

Treatment

➢ General measures:

- Oxygen to maintain $PaO_2 \geq 60$ mmHg or $SaO_2 \geq 92\%$
- IV fluids
- Physiotherapy

➢ Antibiotics:

- Out pt: Amoxicillin + Clarithromycin
- In hospital: 3rd generation Cephalosporins + Macrolide (Erythromycin/Clarithromycin)

CURB 65 score = Poor prognostic factors:

- Confusion
- Urea < 7 mmol/l
- Respiratory rate raised > 30/min
- Systolic BP <90 and/or diastolic BP < 60
- Age > 65

Higher CURB 65 score is associated with poorer prognosis

Note: Organisms associated with high CAP mortality: Influenza A > Strep. pneumoniae > Legionella. Other poor prognostic factors are: Bacteremia, Concurrent alcoholism, and associated CHF or Liver or Renal disease.

Complications of pneumonia

- Respiratory failure type 1
- Para-pneumonic effusion [most commonly with Strep. pneumonia]
- Pneumothorax [Staph. aureus]
- Empyema • Lung abscess • ARDS • Renal failure

DDx of Recurrent pneumonia:

1. Diffuse obstruction [Bronchiectasis, COPD, Asthma]
2. Localized obstruction [Foreign body, Tumor, Lymph node]
3. ↓ Immunity [HIV, Hypogammaglobulinemia, Multiple myeloma]
4. Recurrent aspiration pneumonia
5. Alcoholism

Causes of CAP and their specific feature	
Etiology	*Clinical features*
Streptococcus pneumoniae	Epi: ↑ in Winter. **Commonest cause of CAP** in normal pt, COPD , & DM pt. C/P: Typical pneumonia + **Rusty sputum**. Associated with **Herpes labialis** CXR: Lobar mainly lower lobes. **Dx:** Pneumococcal antigen in Sputum or blood Rx: Benzylpenicillin (the treatment of choice) or Amoxicillin
Mycoplasma pneumoniae	Epi: ↑ in Autumn. More Children. Epidemics every 3-4 years. C/P: Atypical ± Rare complications: 1. Bullous myringitis 2. Erythema nodosum 3. GBS 4.Meningoencephalitis 5. Autoimmune hemolytic anaemia (cold-type) CXR: Bronchopneumonia **WBC**: usually normal **Dx:** IgM for Mycoplasma. Rx: Tetracycline or Macrolide (Erythromycin/Clarithromycin)
Legionella pneumophila	Epi: ↑ in Summer[Epidemics around cooling tower]. Middle to old age. Recent foreign travel. C/P: Atypical ± Diarrhea ± Myalgia. Lab: Hyponatremia, Hypoalbuminemia, Proteinuria, Hematuria.↑ Creatine kinase CXR: Bronchopneumonia or lobar. **Dx:** Legionella antigen in blood or urine. Rx: Ciprofloxacin or Clarithromycin
Staphylococcus aureus	Epi: ↑ in Winter. Often preceded by influenza. C/P: Typical ± **Cavitations**, Hemoptysis. Thin-walled abscess = pneumatoceles. Dx: Gram stain of sputum Rx: Flucloxacillin + Clarithromycin
H. Influenza	More common in COPD pts. **CXR**: Bronchopneumnia Rx: IV cefuroxime
Klebsiella	More in Alcoholics. Affects Upper lobe.
Chlamydia psittaci	Related to contact with birds. Associated with Hepatosplenomegaly

Hospital-acquired pneumonia

Definition: a pneumonia occurring at least 2 days after admission to hospital.

Etiology:

1. *Pseudomonas*
2. *Staph. aureus*
3. *Klebsiella*

C/P, Ix as CAP

Management: Cover for gram –ve microorganisms:

- 3rd-generation Cephalosporin + Aminoglycoside (e.g. gentamicin) OR
- Meropenem

Aspiration pneumonia

Definition: a pneumonia that occurs following large-volume inhalation of gastric or pharyngeal contents.

Epidemiology: common in alcoholic and pt with CVA, or seizures.

Etiology: Usually Anaerobes from mouth flora or Gram-negative organisms from gut

CXR: Lobar consolidation mostly in **right middle** or lower lobe [Rt bronchus is wider & more straight than the Lt bronchus] Lung abscess may occur

Treatment: Amoxicillin+ metronidazole

Lung abscess

Definition: a cavity containing pus in the lung.

Epidemiology: most common in alcoholic men aged >50 years.

Etiology
- S. aureus
- Anaerobes
- Klebsiella pneumoniae
- Legionella pneumoniae

Clinical picture

- Symptoms:
  - Constitutional symptoms [High grade fever, anorexia, malaise]
  - Productive Cough ± Hemoptysis
  - Pleural pain due to pleurisy of the pleura overlying the abscess
- Signs:
  - Halitosis [suggestive of anaerobic infection]
  - Clubbing
  - Resp. Ex: features of consolidation

Investigations

Microbiologic culture from sputum and blood.

CXR & CT: large circular lesion with cavitation and an air/fluid level.

Treatment
- Physiotherapy to drain the abscess
- Antibiotics: Amoxicillin+ Metronidazole for 4-6 weeks

Note: Brain abscess is a rare complication

Pneumonia in the immunocompromised host

↓

Pneumocystis pneumonia (PCP)

Definition: A pneumonia due to infection with Pneumocystis jiroveci (previously termed Pneumocystis carinii). It mainly infects HIV pt.

Clinical features

- Symptoms: Gradual onset of dry cough and exertional dyspnea ±Fever.
- Signs: Chest examination is typically normal. Pt may present with pneumothorax.

Investigations

- CXR: Classically a bilateral perihilar infiltrates that progress to alveolar shadowing.. CXR is normal in 10% of cases. Pleural effusions are very rare.
- CT is done if CXR is normal and may show a bilateral ground-glass pattern.
- ABG: Hypoxia is common.
- Lab: WBC is usually normal. Serum lactate dehydrogenase is typically raised
- Dx is done by seeing the organism in lung or its secretions: Induced sputum microscopy if - ve → Bronchoscopy with BAL which is the diagnostic investigation of choice but if -ve → Lung Biopsy [Gold-standard]

Rx

- Co-trimoxazole [trimethoprim + sulphamethoxazole] IV for 3 Wks is the drug of choice
- High-dose steroids for pts in respiratory failure
- Supportive therapy: O_2 & Fluids

Pulmonary hydatid disease

Overview:

- Hydatidosis is the commonest parasitic lung disease worldwide
- Infection follows ingestion of parasite eggs from dog feces.

Cause: Echinococcus granulosus,

C/P: Cough may have cyst contents, (hydatidoptysis), hemoptysis, & chest pain. The development of fever indicates infection.

Investigations:

- CBC: Eiosinophilia (not in all cases)
- CXR: Rounded cysts mainly in lower lobes ± Water-lilly appearance ± Calcification of the walls. [CT may show daughter cysts]
- Serology: Indirect hemagglutination test (highly sensitive).
  Immunoblot assays for antibodies to antigen arc 5 (specific)

Rx: Best is with surgical excision. But if the pt is unfit for surgery or if the cyst ruptured then treatment is medical with Albendazole.

Tuberculosis [TB]

Definition & Etiology

Tuberculosis (TB) is caused by organisms of the Mycobacterium tuberculosis complex which includes:

1. *M. tuberculosis* (MTB): the most important cause of disease.
2. *M. bovis*: a less important cause of disease
3. *M. africanum*

Modes of Transition:

1. Droplet inhalation infection is the most common way.
2. Ingestion of unpasteurized milk (*M. bovis*)
3. Rare forms of transmission (through the skin, congenitally trough placenta)

Epidemiology

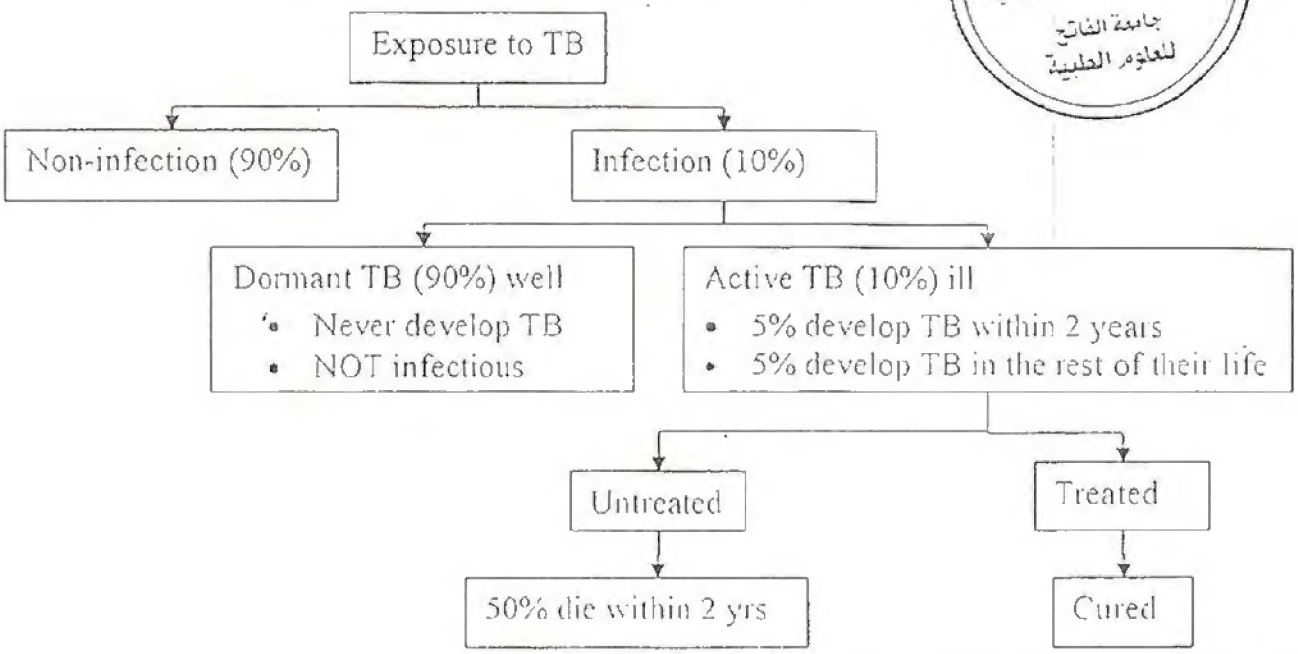
- Worldwide 2 Billion are infected with 8 million new cases of active disease per year.
- TB kills > 2 million yearly, so it is the 2nd leading infectious cause of death after HIV

Q. What is the difference between TB exposure, TB infection, and TB disease?

- **TB Exposure**: it describes an individual who has a **recent contact** with an Pt with **contagious pulmonary T.B.** Clinically, tuberculin skin test, & CXR are all normal
- **TB infection**: it describes an **Asymptomatic** pt with **positive tuberculin skin test** without clinical or CXR signs of illness.
  - Risk factors for infection: Living with pt who has infectious TB is the most important risk factor for acquiring infection.
  - The longer the duration of undiagnosed TB, the greater the severity of disease, and the more intimate the contact, the greater the chance of becoming infected.
- **TB disease = Active TB:** describes a pt with **signs & symptoms of TB ± -ve CXR**
  - Disease may be pulmonary or extrapulmonary. & May be primary or secondary
  - The main determinant for disease is host immunity.
  - Factor ↑ the risk of disease: 1. Old age 2. Alcoholic 3. Silicosis 4. DM type 1 5.Malignancy 6.Malnutrition 7. ↓ immunity (AIDS, Corticosteroid, Inflaximab)

Differentiation between infection and disease		
Criteria	Infection	Disease
PPD+	Yes	Yes
Symptoms	No	Yes
CXR	No	Yes

Natural History for patient with TB



Classification of TB disease

- According to organ involved into Pulmonary and Extrapulmonary
  - In normal person 90% of pts develop pulmonary TB and 10% Extrapulmonary
  - In HIV pt 50% of pts develop pulmonary TB and 50% Extrapulmonary
- According to onset of symptom after infection into:
  - Primary: disease occurs in previously uninfected pt (Tuberculin -ve)
  - Secondary: disease occurs in previously infected pt (Tuberculin +ve)

Pulmonary Tuberculosis

➢ *Primary TB* (children-type)

Definition: infection of a previously uninfected (tuberculin-negative) individual
Age: Children
Pathology & CXR: Primary complex→ Area of consolidation in the Right Middle lobe (Ghon lesion) + Tuberculus lymphadenopathy + Lymphangitis
Note: Cavitations are rare in primary TB.

Enlarged lymph nodes may compress bronchi and lead to	Complete obstruction → Collapse
	Partial obstruction → Emphysema → Bronchiectasis

Clinical features

- Usually asymptomatic infection occurs but in immunosuppressed pts and children, primary infection may progress clinical disease + Pleural effusions.
- Hematogenous spread with Miliary TB or Tuberculous meningitis may occur

➢ *Postprimary* (adult-type)

Definition: disease develops due to reactivation of a latent T.B infection.
Age: Adult
Pathology & CXR: Cavitations are common Usually localized to the Apical and posterior segments of the upper lobes and the superior segments of the lower lobes.
Clinical features

- Constitutional symptoms: Fever, Night sweats, Anorexia, & Wight loss.
- Cough is dry in the first wks & then becomes productive of purulent sputum
- Extensive disease may cause dyspnea.
- ± Hemoptysis, may be massive due to erosion of vessel in the wall of a cavity.

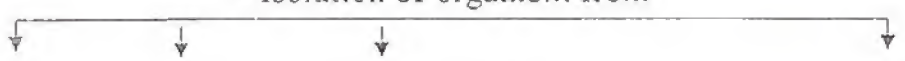
Miliary TB: Miliary TB is due to hematogenous spread of TB. May occur in primary or post primary TB. Pathologically there's yellowish granulomas 1–2 mm like millet seeds

Clinical Features of Miliary TB		
	Classical	Cryptic
Age	< 40 years	> 60 years
Symp	Constitutional symptoms	Constitutional symptoms
Sings	Hepatosplenomegaly + lymphadenopathy Eye → Choroidal tubercles: occur in ⅓ of pt & are pathognomonic. Meningismus occurs in <10%	Hepatosplenomegaly but no lymphadenopathy NO NO
CXR	Miliary pattern	Normal chest X-ray
CBC	Pancytopenia is Rare	Pancytopenia is common
Ix	• -ve tuberculin skin test •Hyponatremia	• Dx Confirmation by biopsy of liver or BM

Investigations (T.B is a bacteriological diagnosis)

1- Bacterial examination:

- Analysis of morning Sputum of 3 days by Ziehl-Nelson stain, if any is +ve the pt is considered as Sputum +ve Tuberculosis
- If sputum is - ve for 3times = Sputum –ve Tuberculosis
- Isolation of organism from



Sputum Child Bronchial lavage Tracheal aspiration

↓

Gastric lavage

- Culture & sensitivity of TB using Lowenstein-Jensen Media takes 4-8 wks to give +ve results but a new broth culture named BACTEC takes 1-2 wks only

2-CXR: (apical lesion)

* Cavity
* Consolidation
* Collapse
* Fibrosis
* Effusion
* Military shadow

3-Tuberculin skin test: (It is an intradermal test)

Positive tuberculin test criteria:

- Greater than 15 mm in a normal host from a low- risk group
- Greater than 10 mm in a high-risk individual
- Greater than 5 mm in an HIV-infected individual

Causes of False-negative Tuberculin skin test:

1. Miliary TB
2. Immunocompromised pts [HIV, Malnutrition, Malignancy]
3. Sarcoidosis
4. 15% of normal patients with TB

Causes of False-positive Tuberculin skin test:

1. Previous vaccination
2. Infection with atypical mycobacteria

4- E.S.R: → ESR ↑↑ in active .T.B. (usually> 100)

→ is used in follow up

→ Can rule out T.B if normal

5-Blood picture:

- WBC: Often Normal or Leucopenia with ↑↑ lymphocytes [± Leukemoid reaction may occur in Miliary TB]
- ↓ Hb: anemia of chronic illness

Treatment

According to WHO TB pts are classified into the following categories:

Cate-gory	Tuberculosis case	Recommended Treatment Schedule	
		Initial phase	Continuation phase
1	➢ New smear +ve Pulm .TB ➢ Severe extrapulmonary TB [All except those in category 3]	2 mo HRZE (SHRZ)	4 mo HR
2	➢ Smear+ve Pulm .TB with : o Relapse [Rx → Smear –ve than becomes Smear +ve again] o Failure [Rx → remains Smear +ve] o Return after interruption	2 mo SHRZE + 1 mo HRZE	5 mo HRE
3	➢ Smear-negative PTB ➢ Less severe extrapulmonary TB [LN, Unilat pleural effusion]	2 HRZ	4 HR

	Anti-TB drugs				
	Isoniazid H	Rifampicin R	Pyrazinamide Z	Streptomycin S	Ethambutol E
Rout	Oral	Oral	Oral	IM	Oral
Mechanisim	Cell wall synthesis Bactericidal	DNA transcription Bactericidal	Unknown Bactericidal	Protein synthesis Bactericidal	Cell wall synthesis Bactericidal
Side-Effects	All may → Hepatitis & Rash				
	Seizures SLE-like synd Peripheral neuropathy	Hemolysis ↓ Plt Orange body secertion	Hyperuricemia Gout	8th nerve damage	Retrobulbar neuritis Peripheral neuropathy

Pt with smear +ve TB should be isolated for 2 week after receiving Anti-TB

Indications of corticosteroids in pt with TB:

1. Tuberculous pleurisy
2. Tuberculous pericarditis
3. Tuberculous meningitis
4. In case of allergy to Anti-TB drugs
5. as replacement therapy after TB-adrenal failure.

BCG vaccine[attenuated *M. bovis*]: protect children from r TB meningitis & Miliary TB

Lung Cancer

Epidemiology

- The most common cause of **cancer death** in men & women [18% of cancer deaths]
- Gender: Male > Female
- Risk factor:
  1. 90% smoking-related [Stopping ↓ risk but it remains higher than in non-smokers]
  2. Asbestos exposure
  3. Arsenic & heavy metal exposure

Types of lung cancer

- Non-small cell lung cancer (NSCLC)
- Small-cell lung cancer (SCLC)

Common types of Bronchial carcinoma		
	[80 %] **Non-small cell lung cancer (NSCLC)** o Squamous [35 %] [Most Common] o Adenocarcinoma [30 %] o Large-cell [15 %]	[20 %]**Small-cell lung cancer (SCLC)** = Oat cell carcinoma It is characterized by early spread to liver, bone, brain, adrenal [Metastasis is usually present at time of Dx]
Rx	Surgery if suitable	No surgery Chemotherapy is treatment of choice ± Radiotherapy
Prognosis	Relatively better	Poorer

Characters of some types:

- **Squamous cell carcinoma** is the commonest type. Usually central & may cavitate and may look like an abscess. This tumor is characterized by the production of parathyroid hormone (PTH)-related peptides → Hypercalcemia.
- **Adenocarcinoma** is not smoking-related, more in females. Can occur in scar tissue or sites of fibrosis. Usually peripheral and they don't cavitate.
- **Large cell cancers** tend to present as peripheral masses, and may cavitate.
- **Alveolar cell carcinoma** = Bronchoalveolar carcinoma is a rare type of adenocarcinoma. It can cause copious sputum production (bronchorrhoea).

Clinical picture

➢ **Local tumor effects**

1. Any respiratory symptom:
   - Persistent cough is the most common symptom
   - Hemoptysis [which may be life threatening]
   - Chest pain (suggests chest wall or pleural involvement)
   - Unresolving pneumonia or lobar collapse
   - Dyspnea (due to bronchial narrowing or obstruction)
   - Wheeze [obstruction below carina]or Stridor [obstruction above carina]
2. Downward invasion → Shoulder pain (due to diaphragm involvement)
3. Lateral invasion →Pleural effusion (due to direct tumor extension or pleural metastases)
4. Medial invasion:
   - Hoarse voice [invasion left recurrent laryngeal nerve]
   - Dysphagia
   - Raised hemi-diaphragm [phrenic nerve paralysis]
   - Superior vena cava obstruction [Lung CA is the commonest cause. It occurs in 10% of pts]
5. Apical tumors = Superior sulcus tumor = Pancoast's tumor:
   • It is NSCLC [Mostly squamous cell or but can be adenocarcinomas] located at the top end of right or left lung and it may cause:
   - **Pancoast's syndrome**: pain in shoulder+ inner aspect of the arm with weakness of hand small muscles due to invasion of the brachial plexus [C5/6, T1].
   - Pancoast's tumor may cause → **Horner's syndrome** (meiosis, ptosis, enopthalmos, anhidrosis) due to invasion of the sympathetic stellate ganglion.

➢ **Metastatic tumor effects**

- Lymph nodes → Lt lung may metastasis to Lt supraclavicular LN [Virchow's LN]
  → Rt lung may metastasis to Rt supraclavicular LN [Scalene LN]
- Liver, Brain, Bone, Adrenal glands

➢ **Paraneoplastic syndromes**

- Clubbing & Hypertrophic pulmonary osteo-arthropathy (any cell type; more common in squamous and adenocarcinoma)
- Nephrotic syndrome
- Peripheral neuropathy
- Erythema gyratum repens
- Dermatomyositis and Polymyositis
- Squamous cell carcinoma is characterized by the production of parathyroid hormone (PTH)-related peptides → Hypercalcemia.

- Paraneoplastic syndromes associated with SCLC:
  1. Syndrome of inappropriate ADH (SIADH).
  2. Ectopic ACTH (Cushing's syndrome).
  3. Eaton-Lambert syndrome.
  4. Cerebellar syndrome.

Examination:

- Normal
- Collapse
- Consolidation
- Pleural effusion

Investigations

- CXR (PA and lateral): may show mass, pleural effusion, rib destruction, intrathoracic metastases, mediastinal lymphadenopathy. CXR can be normal
- PFT for preoperative assessment
- Blood tests: Na^{+}, Ca^{++}, and LFT.

- Investigation to confirm the diagnosis [histological diagnosis]
- **Flexible bronchoscope: useful for central tumors.**
- **Percutaneous needle biopsy under CT or US guide For peripheral tumor.**
- Sputum cytology only indicated in pts who are unfit for bronchoscopy or biopsy
- Pleural biopsy in pts with pleural effusions.
- Mediastinoscopy, Thoracoscopy or Thoracotomy may be needed.

- Investigations for stating
- CT chest for mediastinal LN, CT abd. for Liver & Adrenals assessment.
- Bone scan if pt has bony pain, ↑ Ca^{++}, ↑ alkaline phosphatase.

Contraindications for surgical resection:

1. [T4]: Invasion of mediastinal structures : Heart[cardiac tamponade], Great vessels [SVCO]; Nerve invasion [vocal cord palsy or phrenic nerve palsy]; Trachea & Esophagus, Malignant pleural effusion.
2. Contralateral mediastinal nodes [N3]
3. Distant metastasis [M1]
4. $FEV_1 < 0.8$ liters
5. Severe or unstable cardiac or other medical condition

Note: Paraneoplastic features are NOT contraindications for surgery

Management

- Surgery: if respectable 5-yrs survival rates is 75% in stage I disease and 55% in stage II disease.
- Chemotherapy: In SCLC use Chemotherapy ± Radiotherapy, can ↑ survival a from 3 months to 1 year.
- Radiotherapy: less effective than surgery but may improve survival.

Prognosis

Prognosis is poor → 20% surviving at 1 yr and 5% at 5 yrs

Topics related to lung cancer

Lymphangitis carcinomatosa

Definition: it is the infiltration of pulmonary lymphatics by tumor.

Causes: 1. Lung CA
2. Breast CA
3. Prostate CA
4. Stomach CA
5. Pancreatic CA

C/P: Causes dyspnea, cough, and systemic signs of advanced malignancy.

Ix: CXR → Fine linear shadows throughout both lung fields.

Rx: Oral steroid treatment and diuretics can give symptomatic relief. Prognosis is poor

Hypertrophic pulmonary osteo-arthropathy

Definition: it is a disease characterised by periostitis of distal tibia fibula, radius and ulna.

Causes: Same causes of clubbing

C/P: Pain and tenderness over the affected bones with pitting edema over the shin.

Ix: X-rays → subperiosteal new bone formation.

Superior vena cava syndrome

C/P: ↑JVP with absent pulse wave with edema affecting the face, & arms.

Most common cause is lung cancer

Rx: Radiotherapy and Corticosteroids

Deep Vein Thrombosis & Pulmonary Embolism

Introduction

- > 80 % of PE is a complication of DVT in the legs.
- The majority of pts with PE remain undiagnosed.

	Causes of Venous Thromboembolism	
Virchow's triad	*Stasis:* • Immobilization (Stroke, Heart Failure, Old age, Travel) • Obesity • Pregnancy *Vascular injury:* • Trauma • Surgery • Venous catheters *Hypercoagulability:* • Malignancy • Oral CCP & Pregnancy • Smoking • Nephrotic syndrome	*Hereditary Hypercoagulability:* • Factor V Leiden mutation • Protein C deficiency • Protein S deficiency • Antithrombin III deficiency

In history of pt with DVT or PE look for the following:

- In PMH: 1. H/O Trauma
  2. H/O prolonged immobility
  3. H/O previous DVT
  4. Drug history
  5. H/O malignancy [Trousseau's syndrome = migratory superficial thrombophlebitis + CA usually pancreatic]
- SH: Smoking
- FH: +ve family history.

DVT Clinical picture

Symptoms

- **Calf pain** [commonest symptom] ± Unilateral leg swelling, Redness, Hotness Fever

Signs

- **Calf asymmetry [unilateral lower limb edema]** [Asymmetry of >1cm is significant, measured 10 cm below the tibial tuberosity]
- Erythema & Hotness
- Tenderness [In calf vein DVT over the back of calf ms. In Femoral vein DVT it is over the antero-medial side of the thigh].
- **Homans' sign** (calf pain with resisted dorsiflexion of the foot)
- Dilated superficial veins → palpable cords in the posterior calf

PE Clinical picture

Symptoms

- **Sudden dyspnea** is the most common symptom.
- Cough
- Pleuritic chest pain
- Hemoptysis [occurs in 10-15% of pts]
- Syncope

Signs

- **Tachypnea** the most common sign.
- Tachycardia
- Pulmonary emboli → pulmonary hypertension → RVF
- Chest exam: usually normal but plural rub is heard in pulm infarction.

DDx of DVT:	DDx of PE:
1. Cellulitis.	1. Pleurisy.
2. Venous insufficiency.	2. Pneumothorax.
3. Ruptured Baker's cyst.	3. MI
4. Lymphedema.	4. Pericarditis.
5. Muscular injury.	5. Congestive heart failure.

Massive PE
- → *Definition*: PE + hemodynamic instability (BP<90mmgh).
- → *Cause*: Bilateral thrombi affecting ≥ 50% of pulmonary circulation.
- → *C/P*: Mainly Dyspnea ± Syncope; Chest pain is unusual.

Pulmonary Infarction: Occurs in <10% of PE & is suggested by finding:

- Hemoptysis
- Infarction → pleurisy → chest pain & plural rub on auscultation
- Infarction may → atelectasis → elevation of hemidiaphragm in the affected side
- Bloody pleural fluid on aspiration

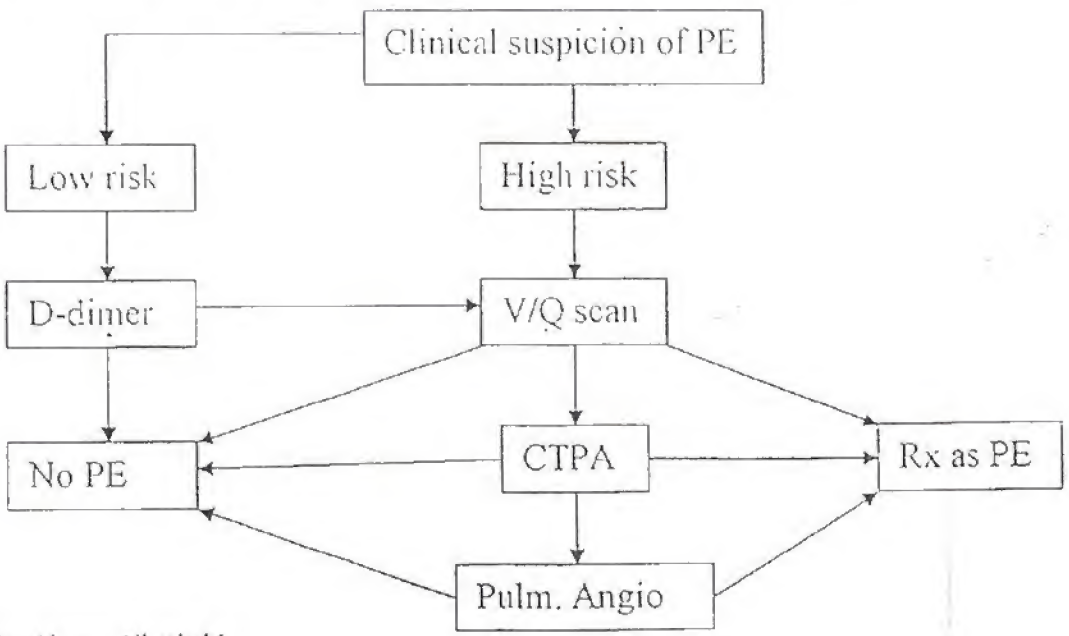
Investigations

➢ DVT

- **Duplex venous ultrasonography is the most commonly used test to confirm Dx.**
- **Contrast venography is the gold standard** but is done if the US is equivocal.
- D-dimer (a fibrin degradation product) is very sensitive but its not specific.

➢ PE

1. D-dimer (a fibrin degradation product) if normal the PE is very unlikely
2. ABG: ± hypoxemia ± respiratory alkalosis.
3. ECG may show:
   - Most frequent ECG change is T wave inversion in the anterior chest leads
   - Sinus tachycardia • RAD [Right axis deviation] • RBBB [Right bundle branch block]
   - S1-Q3-T3 [Deep S wave in lead I and a deep Q wave and T wave inversion in lead III, this change is specific but not sensitive presenting only in 10% of pts]
   - Pseudo-infarction pattern or Strain pattern in the inferior leads (II, III, aVF).
4. Chest X-ray is frequently normal but may show:
   - **Westermark's sign**: decreased vascular markings in the affected lung regions.
   - **Hampton's hump**: a wedge-shaped peripheral infiltrate.
   - **Palla's sign**: an enlarged right descending pulmonary artery
5. **Ventilation/perfusion (V/Q) lung scan is the usual initial diagnostic test for PE.**
6. CTPA [CT of the pulmonary arteries with intravenous contrast].
7. **Pulmonary angiography [Gold standard].** But done only if other test are equivocal



Treatment of PE

- As all ER Call for help
- Stabilize the pt: ABC
  - Give high concentration O2 to correct hypoxia
  - Insert 2 IV cannula and start infusion of crystalloid or colloid
- Analgesia with oral NSAID [Opiate better avoided due to its vasodilator effect may → ↓BP
- Anticoagulation:
  - In pts with suspected DVT or PE, anticoagulant therapy should be started immediately.
  - **Heparin** Unfractionated or Fractionated[therapeutic range is 1.5-2.0 times the control PTT] followed by **Warfarin** oral with overlap with heparin for at least 4–5 days until Warfarin therapeutic range is reached [INR 2-3] then continue with Warfarin only.
  - Duration of Warfarin therapy is 3 months in DVT & 6 months in patients with PE.
    - Unfractionated heparin dose: 7500- to 10,000-U bolus, followed by continuous IV infusion of about 30,000-40,000-U/day (about 1500-2000 U/hour)
    - Fractionated heparin dose: Enoxaperin 1 mg/kg SC bid.
  - Thrombolytics are used Only in Pts with massive PE. If contraindicated then inferior vena cava filter is used.

Treatment of DVT: Anticoagulation treatment as PE

DVT prophylaxis

- Non-pharmacological:
  - Prevention of dehydration by giving adequate amount of fluid
  - Mobilization of the patient
  - Elastic stocking
  - External intermittent pneumatic compression.

- Pharmacological:
  - Low dose S/C unfractionated heparin [5000 U twice daily] or LMWH [e.g., enoxaparin 30mg twice daily.

Q. How can DVT cause stroke?

Q. How long it takes for DVT to develop following fracture or surgery?

Q. How to differentiate clinically between DVT and Cellulitis?

DVT	Cellulitis
Tenderness localized over veins	No localized tenderness over veins
Less reddening	More reddening
No portal of entry	Portal of entry is usually present

Pneumothorax [PTx]

Definition: The presence of free gas in the pleural space.

Epidemiology

- Gender: Male > Female
- Primary spontaneous type is more common in young [more in Marfan pt]
- Secondary spontaneous type is more common in old

Etiology

- Spontaneous PTx: occurs without H/O thoracic trauma it is 2 types:
  1. **Primary spontaneous PTx**: occurs in the absence of lung disease & is due to rupture of a congenital bleb. More in Young (15-30 yrs) Tall, Smokers.
  2. **Secondary spontaneous PTx**: it occurs in pts with pre-existing lung disease (Asthma, COPD, Bronchiectasis, TB, Pneumonia).
- Traumatic: pneumothorax following trauma either blunt or penetrating

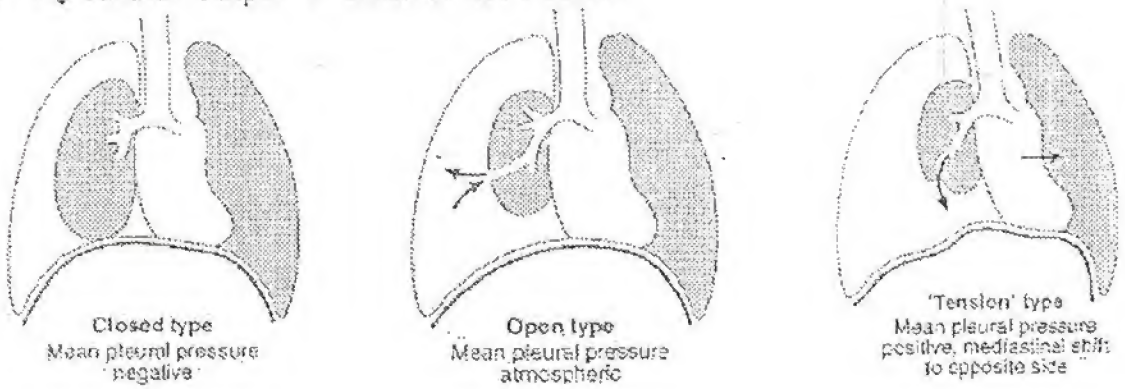
Pathophysiology

The effects of a pneumothorax depend on whether the pleural leak persists or not:

- Closed PTx: the communication between the lung and pleural space is closed → no more leak, Pleural pressure remains negative & resolution will occur even without Rx

- Open PTx: there is persistent communication between the airway and pleural space (bronchopleural fistula) → persistent leaking. [i.e. bubbling of the chest drain]. Here air enters and gets out of pleural space. Pleural pressure = Atmospheric pressure. Pt has high risk of infection (empyema).

- Tension PTx: occurs when the leak remains open and acts as a one-way valve between the airway and pleural space → progressive ↑ of air in the pleural space→ Positive pleural pressure [pleural pressure > atmospheric pressure] → compression of the underlying and contralateral lung with heart and mediastinal shift → kinking of IVF →↓ cardiac output → death If not treated.



Clinical manifestation

- May be asymptomatic if small.
- Sudden Chest pain +Dyspnea is the most common presentation.
- **If Tension PTx** it will → Tracheal deviation to opposite side, ↑ JVP, ↑ HR, & ↓ BP
- **Subcutaneous emphysema** can occur from air leak into skin & subcutaneous tissues, felt as a crackling sensation in skin. May→Facial swelling & airway obstruction.

Resp. Ex.

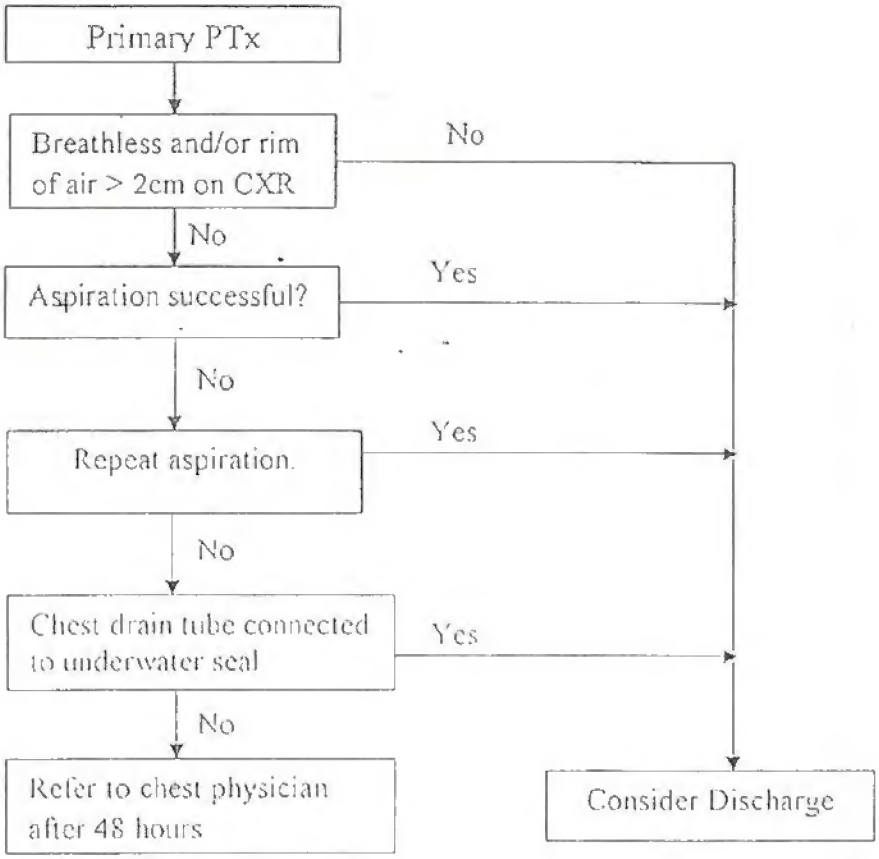
- *Inspection*
  - ↑ RR
  - ↓ chest expansion on the affected side
  - ± Deviation of mediastinum to opposite side
- *Palpation*
  - ↓ chest expansion on the affected side
  - TVF ↓ on the affected side
- *Percussion*
  - **Hyperresonant** on the affected side
- *Auscultation*
  - ↓ Air entry unilaterally
  - Vesicular breathing
  - No added sound

Absent breath sounds + resonant percussion = pneumothorax

Investigations

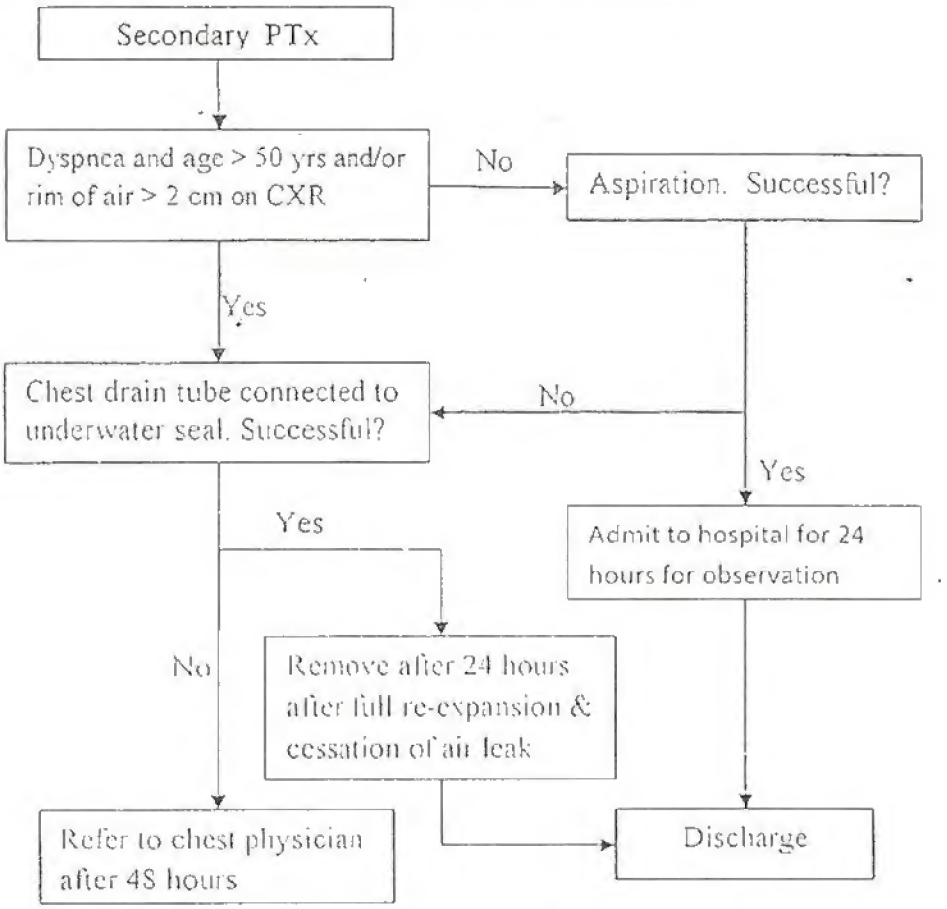
- ABG & Pulse oxymetry: may show hypoxemia.
- **CXR** is diagnostic by showing black shadow (loss of lung markings) with lung collapse. [If there is doubt between large emphysematous bulla and pneumothorax do CT to differentiate]. Mediastinal deviation suggests the presence of tension.
- Ultrasonography or CT are both superior to CXR for detection of small PTx

Management of Primary pneumothorax



- If the rim of air is < 2cm & pt is not dyspneic → discharge after 6 hrs of observation
- If the rim of air is > 2cm &/OR pt is dyspneic then aspiration should be attempted
- If aspiration fails then repeat aspiration should be considered
- If 2nd aspiration fails then a **Chest drain connected to underwater seal**:
  - The drain is inserted on the 5th intercostal space in the mid-axillary line
  - If the lung has re-expanded and the drain is not bubbling, clamp the tube and wait for 2 hours –then→ repeat CXR if no PTx –then→ remove the drain & keep the pt admitted for 24 hours –then→ repeat CXR to exclude recurrence & then discharge
  - Continued bubbling after 5-7 days is an indication for surgery
- Airplane travel: pts with PTx should not fly for 3 months because the pressure changes can lead to expansion of the gas in pleural space → tension PTx.

Management of Secondary pneumothorax



- If the pt is < 50 yrs & the rim of air is < 2cm then attempt one aspiration and If aspiration fails a chest drain should be inserted
- If the pt is dyspneic & is > 50 yrs OR the rim of air is > 2cm then a Chest drain connected to underwater seal should be inserted. [as primary PTX]
- All patients should be admitted for at least 24 hours

Management of tension pneumothorax [EMERGENCY]

- Tension pneumothorax is a clinical diagnosis
- Never leave the pt unattendent [for e.g. sending the pt to CXR]
- Give maximal oxygen to reverse hypoxia
- Insert the largest cannula available perpendicular to the chest wall into 2nd intercostal space at mid-clavicular line on the side of pneumothorax on examination
- Insert chest drain connected to underwater seal as soon as possible

Prognosis

- After primary spontaneous PTx, 30% of pts have a recurrence within 5 yrs.
- After a second attack, recurrence rate is 50% & Surgical pleurodesis is recommended.

Pleural effusion

Definition: accumulation of fluid in the pleural space.

Etiology and Pathophysiology

A pleural effusion can be classified either as a transudate or an exudate:

- The formation of a transudate reflects disturbance in the Starling forces. The pleural capillary endothelium are intact and the protein content of the fluid is low.
- The formation of an exudate implies a loss of integrity of the pleural membrane and/or disruption of the lymphatic drainage. Which is characterized by ↑ protein.

Causes of pleural effusion
Transudate are caused by: • Congestive heart failure (most common cause of transudative pl-effusion) • Cirrhosis • Nephrotic syndrome • Pulmonary embolism (25%) • Myxedema
Exudates are caused by: • Bacterial pneumonia (most common cause of exudative pl-effusion) • TB • Malignancy, most commonly lung, breast, and lymphoma • Pulmonary embolism (75%) • Collagen vascular diseases (RA, SLE) • Pancreatitis

Clinical picture

- The only symptom is dyspnea.
- Note: effusion of <500ml are difficult to detect clinically.

Resp. Ex.

- *Inspection* → ↑ RR; ↓ chest expansion on the affected side; ± Deviation of mediastinum to opposite side
- *Palpation* → ↓ chest expansion on the affected side; TVF ↓ on the affected side
- *Percussion* → Stony dull on the affected side
- *Auscultation* → ↓ or absent Air on the affected side; No added sound

Investigations

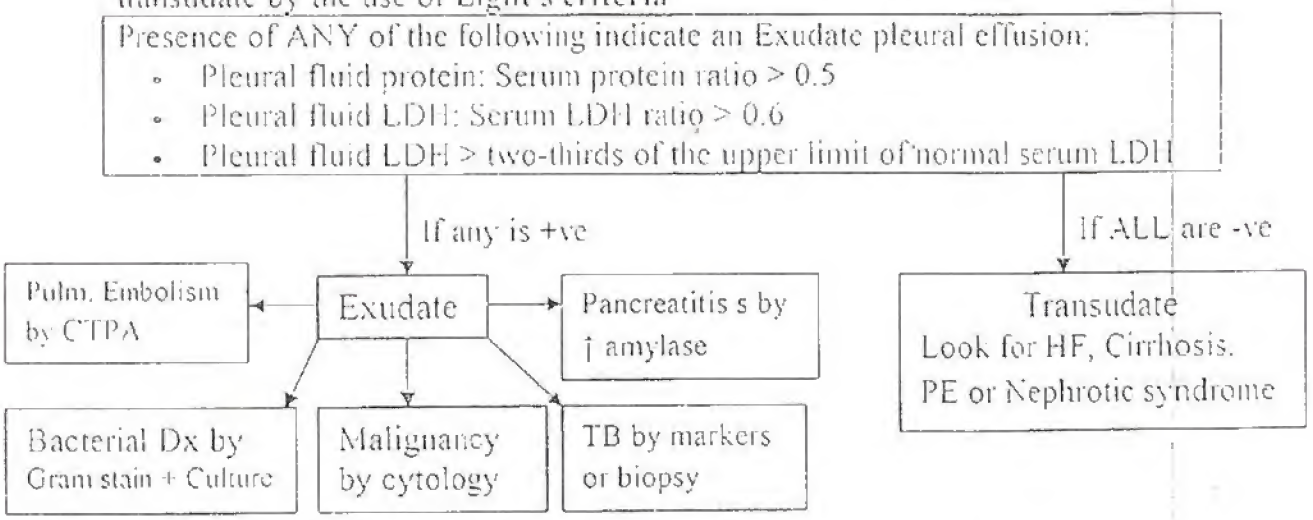
- **CXR**
  - At least 200 ml is accumulated for CXR to show changes. It shows: Homogeneous white shadow of the lower chest with obliteration of the costophrenic angle & hemidiaphragm, upper margin of the opacity is concave & is higher laterally than medially [**Meniscus sign**]
  - Small effusions can be seen by using bilateral lateral decubitus CXR, or CT or US.
  - If the pt is female in reproductive age → ultrasound should be used not CXR

- **Pleural aspiration** [Diagnostic Thoracentesis]

Macroscopic Examination of pleural fluid	
Clear	• Transudate
Straw colored	• TB
White	• Pus[empyema] • Chylous or Pseudochylous
Bloody	• TB • Malignancy • Pulmn. infarction [PE]

- Send Pleural fluid for:
  - Biochemistry: Total Protein, LDH, Glucose, PH, Amylase, TB marker
  - Gram stain, Culture
  - Cytology

- Measure Serum Protein & LDH to and use the results to determine if it is an exudate or transudate by the use of Light's criteria



Interpretation of other test results:

- ↓ Glucose: • Bacterial infection • Malignancy • Very low [Rheumatoid arthritis]
  ↑ Amylase: • Pancreatitis • Esophageal rupture • Malignancy

Note: Other features of Exudate effusion: • Protein > 3gm % • Sp. gravity> 1018 • LDH > 200 IU/L

- **Pleural biopsy** [by Abram needle] is useful for the diagnosis of TB and Malignancy

Parapneumonic effusion: it's defined as an effusion that develops over lung abscess, pneumonia, or bronchiectasis.

Indications of chest tube drain in parapneumonic effusion:

1. Loculated pleural fluid
2. Pleural fluid pH < 7.2
3. Pleural fluid glucose (<60 mg/dL)
4. Positive Gram stain or culture of the pleural fluid
5. The presence of gross pus in the pleural space [empyema]

Tuberculus pleural effusion: Fluid is Exudative with predominant lymphocytosis; bacilli are rarely seen on smear, & culture is +ve in < 20%; so biopsy required for Dx. Pleural fluid TB markers are: • ↑ Adenosine deaminase (ADA) • ↑ Interferon γ

Causes of milky colored pleural effusion			
	Chylous pleural effusion	Pseudochylous	Empyema
Centrifugation	No change	No change	Changes to cells & fluid
Ix	TGA > 110 mg	Cholesterol > 200	-
Causes	1. Mediastinal Lymph node 2. Lymphatic duct trauma 3. Filariasis	1. Rheumatoid arthritis 2. TB	Infection

Treatment of pleural effusion

- Treat the underlying cause
- Therapeutic aspiration is used to relieve dyspnea but removing more than 1.5 liters in one episode is not recommended due to the risk of re-expansion pulmonary edema
- In case of malignancy pleurodesis with tetracycline or talc may be required

Miscellaneous conditions

Acute Respiratory Distress Syndrome [ARDS]

Definition

ARDS it is a severe acute lung injury due to many causes & it is characterized by:

- Acute, persistent, lung inflammation, increased vascular permeability
- Bilateral and extensive infiltrates on the CXR
- PaO_2/FiO_2 <200 mmHg
- Pulmonary capillary wedge pressure of < 18 mmHg (non-cardiogenic)

Pathophysiology

Inflammatory damage to the alveoli, either by locally produced pro-inflammatory mediators, or remotely produced and arriving via the pulmonary artery.

Etiology

- Sepsis/pneumonia; secondary risk factors for developing ARDS, when septic, are alcoholism and cigarette smoking
- Gastric aspiration
- Trauma [Fat embolism]
- Burns
- Blood transfusion
- Acute pancreatitis
- Near drowning

Management

- Treat the underlying cause.
- Supportive care with adequate oxygenation, Mechanical ventilation almost always required to maintain oxygenation.
- High-dose steroids [controversial]

Causes of Hypoxemia

1. ↓ in inspired O2 [high altitude]
2. Hypoventilation [Center or Muscle disease]
3. Ventilation/Perfusion mismatching → ↓ Ventilation [Obstructive airway diseases]
   → ↓ Perfusion [Pulmonary embolism]
4. Diffusion problem: ILD
5. Shunting: Blood goes from the heart Rt side the Lt side bypassing the alveoli and it may by intracardiac [ASD,VSD,PDA] or intrapulmonary [hepatopulmonary syndrome]

Mechanical ventilation

Indications:

1. Respiratory rate > 35 breaths/min
2. $PaO_2 < 60$
3. $PaCO_2 > 60$

Complications: • Pneumothorax • Infections

Pulmonary Hypertension

Definition: mean pulmonary artery pressure (PAP) > 25 mmHg.

Etiology

- Primary Pulmonary hypertension [Rare, occurs in young ♀, poor prognosis]
- Cardiac disease: Mitral stenosis, LVF or Congenital diseases [ASD, VSD, PDA]
- Chronic hypoxemia: [Ch. bronchitis, Bronchiectasis, ILD]
- HIV
- CTD: [Mainly scleroderma]

Clinical features: History: progressive Dyspnea ± Chest pain.
Examination: ↑JVP , Lt parasternal heave[RVH], Loud P2

Dx: Echo or Cardiac catheterization

Rx • Oxygen. • Lifelong Warfarin. • Rx heart failure
• Some pt respond to vasodilators CCB or prostaglandins epoprostenol (prostacyclin)

Pulmonary syndromes associated with *Aspergillus* sp

- **Allergic bronchopulmonary aspergillosis** [ABPA]: occurs in asthma pt.
- **Aspergilloma [Fungus ball]** results from colonization of aspergillus in preexisting pulmonary cavity [pt with previous TB].
- **Invasive aspergillosis**: occurs in pts with severe pancytopenia as AIDS pt

CHEST-X-RAY Interpretation Check list

I. The X-ray	Yes	No
Type of X-ray (plane or contrast)		
Name of patient and Date of the X-ray		
Projection (PA, AP, or lateral)		
Orientation		
Penetration-		
Rotation-		
II. Intervention: Note any chest drains, ECG pads, etc., that may be visible on the X-ray		
III. Looking for Abnormalities		
1-Lung fields		
2-Mediastinum and Trachea		
3-Heart		
4- Pleura and Diaphragm		
5-Chest wall (soft tissue and bone)		
6-look at area under the diaphragm		

Type of the X-ray: The X-ray is divided into 2 types:

1- In Plain X-ray: no contrast media is used.
2- In Contrast X-ray: a contrast media is used.

Projection

- It is the direction of X-rays in relation to the chest and it may be AP, PA, or Lateral
- AP [anteroposterior] the X-ray beam is fired in front of the patient and is taken when the patient can't move from his bed as in emergency situation.
- PA [posteroanterior] the X-ray beam is fired from behind of the patient.
- Lateral the X-ray beam is taken from the side of the patient

Q- How to differentiate between the AP and PA?

1- AP is marked by the radiologist but PA not marked
2- If most of scapulae overlie the lung fields it's AP, and if only the edge of the scapulae or scapulae are outer to lung fields its PA

Q- What is the importance of knowing if the CXR is AP or PA?

1- AP is usually taken in seriously ill patient e.g. pts in ICU.
2- Importance is that heart size will look bigger in AP and you can't comment if it's enlarged or not. So comment on the heart size only in PA.

Orient the X-ray

Orientation is to put the CXR in correct position as if the pt is standing in front of you.

Q- How can you know right from left?

Always First look for the mark made by the radiologist if not comment on its absence, and put the x-ray in correct orientation.

Right side	Left side
1- Right diaphragm is higher 2- Liver shadow	1- Aortic knuckle 2- Cardiac apex shadow 3- Air bubble in the stomach

Rotation: to see if the CXR is centralized or not.

- CXR is will centralized if the distance from the vertebral spine to the medial ends of clavicles is equal
- If CXR is not centralized you cannot comment on tracheal or mediastinal shift or cardiac size

Penetration

- If the dose of the X-ray is less than required it will not penetrate will and it will be a Soft CXR i.e. showing the soft tissues mainly [fatty tissue] and the lung fields will appear whiter.
- If the dose of the X-ray is more than required it will penetrate body tissues and will show the hard tissues [bones]

Q- How to know if it's Good, Soft, or Hard CXR?

Well penetrated→ thoracic spine is just seen behind the heart
Underpenetrated = Soft→ you can't see thoracic spine behind the heart
Overpenetrated = Hard→ thoracic spine is clearly visible behind the heart

Degree of inspiration

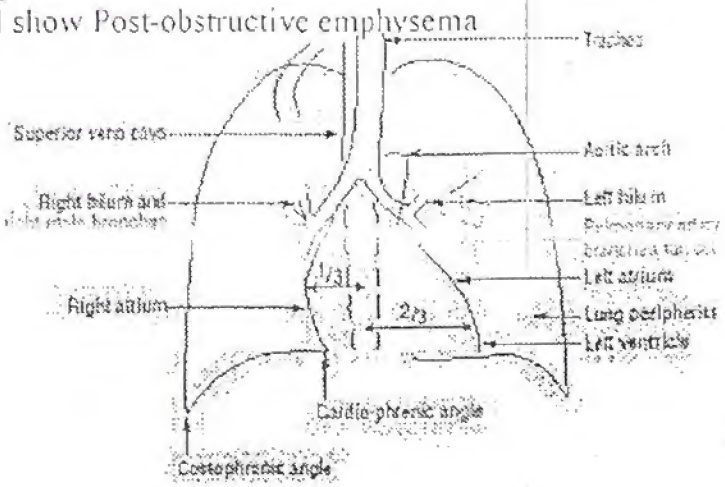
- Ideally CXR is taken in full inspiration in which normal chest the 6th rib will be above the diaphragm anteriorly and 10th rib will be above the diaphragm posteriorly → If > 6th rib ant and 10th rib post the chest is **Hyperinflated**
- Anterior ribs are less dense, more oblique don't reach midline.
- Posterior ribs are denser, more horizontal and reach the midline.

Q- What are the indications of CXR in expiration?

1- FB aspiration→ on expiration it will show Post-obstructive emphysema
2- Small pneumothorax

Looking for abnormalities

1. Lung fields
2. Mediastinum and Trachea
3. Heart
4. Pleura and Diaphragm
5. Chest wall soft tissue & bone

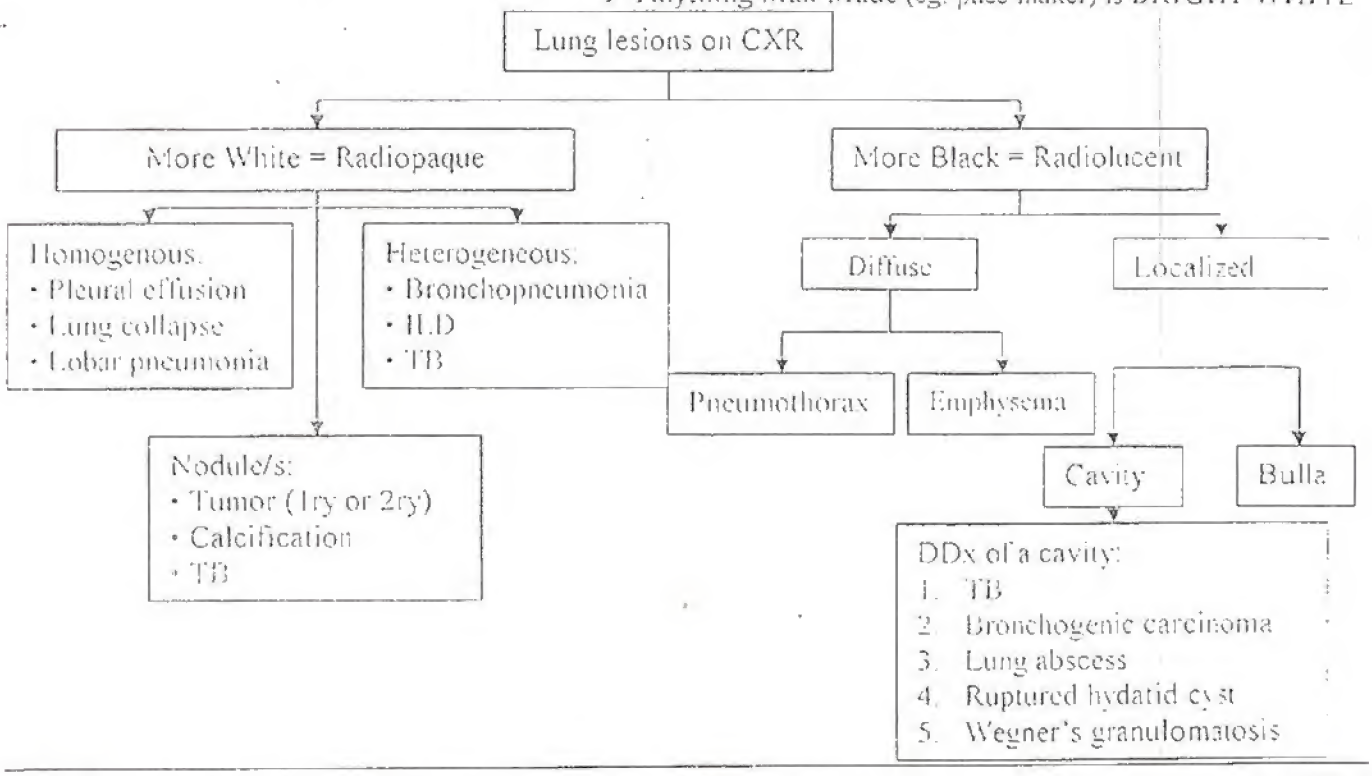


Lung fields: For CXR description the lung is divided into 3 zones:

- Upper zone above the 2nd ant rib
- Middle zone between 2nd and 4th ant ribs
- Lower zone under the 4th ant rib

Compare between lung zones

CXR as a contains *different shades*
- → Bone is WHITE
- → Gas is BLACK
- → Soft tissue is GREY
- → Anything Man-Made (eg. pace-maker) is BRIGHT WHITE



CXR in Pleural effusion: Homogeneous white shadow of the lower chest with obliteration of the costophrenic angle and the hemidiaphragm, upper margin of the opacity is concave to the lung and is higher laterally than medially (meniscus sign)

CXR in Emphysema:
1. Lung is more black
2. Ant ribs are more horizontal
3. Widening of intercostal spaces
4. The lung extend below the 6th rib ant and 10th rib post
5. Heart is tubular
6. Diaphragm is flat

CXR in Pneumothorax:
Absent lung markings peripherally with visible lung edge with Lung collapse

Honeycomb appearance: • Bronchiectasis (hyperinflated chest) • ILD (small lung)
Localized opacity with fluid level: • Lung abscess • Ruptured hydatid cyst